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Table of Contents.

POLIOMYELITIS: AN ACCOUNT OF 90 CASES, WITH REFERENCE TO THE ROLE PLAYED BY TRACHEOTOMY IN BULBAR INVOLVEMENT.

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EPIDEMIOLOGICAL CONSIDERATIONS.

Topographical Incidence.

Of the 90 patients admitted to the Metropolitan Infectious Diseases Hospital, Northfield, from September 1, 1947, to March 6, 1948, 39 (43%) were from rural areas and 51 (57%) from urban areas; in 20% of the rural and 25·5% of the urban cases the disease was of the bulbar type. The majority of the rural cases occurred slightly later than the urban cases, which suggests that the disease began in the city and was later conveyed to the country areas. Figure I illustrates this point well: as patients were admitted to hospital at different stages of the disease, the onset of the disease in each case was taken as being from the beginning of the prodromal stage.

Seasonal Incidence.

The number of patients with poliomyelitis admitted to this hospital each month is shown in Table I.

These figures are relatively similar to those in 1937–1938 in this State, when most cases occurred in December, 1937, and January and February, 1938. This tendency for the majority of cases to occur in the summer months suggests the possibility of the disease's being insect-borne.

Age Incidence.

Table II shows the relative age incidence in the present series compared with the 1937–1938 outbreak.

The youngest patient was a male baby, aged eight months, suffering from moderate paresis of one leg. The oldest was a woman, aged forty-nine years, suffering from mild paresis of the dorsiflexors of one foot.

Table II shows that in this outbreak the disease affected the higher age groups more than in 1937–1938: 35% of the patients were below the age of ten years, compared with 53% below this age in 1937–1938. A noticeable feature of the present series of cases was that the most severe were included in the higher age groups, 76% of the patients with bulbar involvement being aged ten years or over. Also most of the children below this age had relatively mild paralysis. From these figures it can be concluded that in this State at any rate the term "infantile paralysis" is indeed a misnomer.

The number of patients with bulbar involvement in the different age groups is shown in Table III.

Sex Incidence.

From Table IV it can be seen that male subjects were affected slightly more than females, and these figures are similar to those of the 1937-1938 epidemic. Of the patients with bulbar involvement, 62% were males and 38% females.

Familial Incidence.

Two instances occurred of more than one member of a family being affected. In one family, a boy aged eleven years was admitted to hospital on December 30, 1947, suffering from moderate paresis of one leg, and his seventeen-year-old sister was admitted to hospital on January 4, 1948, with paresis of both legs. In the other

family, a girl, aged eleven years, was admitted to hospital on October 21, 1947, with severe paresis of both arms and the spine flexors, and her sixteen-year-old sister was admitted to hospital on February 2, 1948, with an abortive attack of poliomyelitis, the cerebro-spinal fluid containing 40 lymphocytes per cubic millimetre and 30 milligrammes of protein *per centum*.

It is possible that in such examples there is an hereditary constitutional susceptibility to the disease.

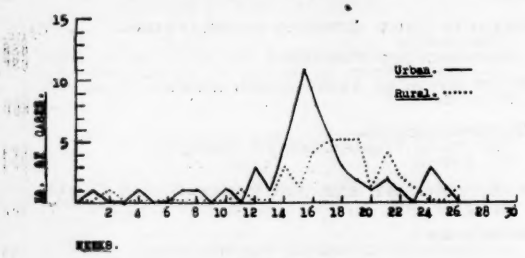

FIGURE I.

Other Factors.

Other factors of interest are as follows.

1. Susceptibility to the disease of patients having recently undergone tonsillectomy. One patient with severe bulbar involvement, a girl, aged eleven years, had had her tonsils and some teeth removed twelve days before her admission to hospital.

2. Relation of the severity of the disease to exercise in the meningeal phase. The histories of a number of the patients with spinal involvement suggested that any undue exercise in the meningeal phase tends to increase the severity of the ensuing paralysis (Russell, 1947).

3. Relationship of pregnancy to the disease. Three patients were pregnant; two of these were in the first four months of pregnancy and the other was only two weeks off full term. Two of these had mild infections, but the other, a woman, aged nineteen years, who was three to four months pregnant, had severe paralysis of both legs.

TABLE I.

Month.	Number of Cases.	Bulbar Involvement. (Number of Cases.)
September, 1947 ..	2	—
October, 1947 ..	2	—
November, 1947 ..	4	—
December, 1947 ..	41	10
January, 1948 ..	29	9
February, 1948 ..	11	1
March, 1948	1	1

CLINICAL FEATURES.

The Onset.

The onset in most cases was fairly acute; particularly was this so in bulbar involvement, which progressed extremely rapidly. Four types of reaction were observed in the prodromal stage, either alone or in combination: (i) meningism—headache and neck and spine rigidity; (ii) coryza—rhinitis, tonsillitis, fever; (iii) pain in affected muscles preceding paresis; (iv) gastro-intestinal disorders—vomiting, constipation, anorexia, colicky abdominal pain, diarrhœa. In the majority of cases there was some degree of combination of all of these. Constipation was another particularly common symptom, only six patients complaining of diarrhœa. The length of the prodromal stage varied from one to nine days, averaging three to four days. No correlation was observed between the type or severity of the prodromal stage and the site, degree or clinical course of the ensuing paralysis. In only one case could no prodromal symptoms at all be elicited; the patient was a woman, aged thirty-four years, who woke up one morning to find her legs powerless.

The "dromedary" type of prodrome, in which an initial coryzal stage is followed by a latent period with abolition of symptoms lasting one to two days, which in its turn is followed by meningeal and perhaps paralytic stages, was observed in 18 cases.

TABLE II.

Age Group. (Years.)	1947–1948. Number of Cases.	1947–1948. Percentage.	1937–1938. Number of Cases.	1937–1938. Percentage.
0 to 4 ..	10	11·0	79	23·2
5 to 9 ..	22	24·5	103	30·2
10 to 14 ..	17	18·5	69	20·2
15 to 19 ..	14	15·5	45	13·2
20 to 24 ..	9	10·0	18	5·3
25 to 29 ..	8	8·9	8	2·4
30 to 34 ..	5	5·6	4	1·2
35 to 39 ..	2	2·2	5	1·5
40 to 44 ..	—	—	4	1·2
45 to 49 ..	3	3·3	2	0·6
50 to 54 ..	—	—	3	0·9
55 to 59 ..	—	—	1	0·3

Paralytic Stage.

The following four main clinical types of paralysis were recognized: (i) spinal, 76 cases (56 pure spinal, 20 bulbo-spinal); (ii) abortive, 12 cases; (iii) encephalitic, 10 cases; (iv) bulbar, 21 cases.

These types occurred alone or in combination; in all except one of the bulbar cases, and also in all except one of the encephalitic cases, evidence of spinal involvement was found in addition. These four main groups will now be considered separately.

TABLE III.

Age Group. (Years.)	Bulbar Paralysis. (Number of Cases.)	Percentage.
0 to 4	—	—
5 to 9	5	24
10 to 14	7	33
15 to 19	4	19
20 to 29	4	19
30 to 44	—	—
45 to 50	1	5

Spinal Paralysis.

Of the 76 cases of spinal involvement, in 25 paresis of neck flexors was present, in 14 paresis of one arm, in 13 paresis of both arms, in 16 paresis of intercostal muscles, in 15 paresis of the diaphragm, in 18 paresis of abdominal muscles, in 10 paresis of one leg, in nine paresis of both legs and in 12 paresis of both arms and legs.

TABLE IV.

Sex.	Number of Subjects.	Percentage.	1937–1938 Percentage.
Male	52	57·8	60
Female	38	42·2	40

Retention of urine occurred in seven cases, in all of which paralysis of one or both legs was present; five of these patients were passing urine normally after two or three days, but the other two required regular catheterization for seven to eight days before return of function.

Constipation occurred in all but two of these cases. These two patients suffered from nausea and diarrhœa in

the prodromal stage. This high incidence of constipation suggests some involvement of the autonomic nervous system.

Mortality.—Two deaths occurred. The first was that of a male, aged 21 years, and was due to acute ascending paralysis with respiratory and probably circulatory involvement. The other was that of a male, aged thirty-eight years, and was due to a massive pulmonary embolus occurring eleven days after the onset of paralysis.

Treatment.—Treatment consists essentially of complete muscular rest during the acute phase, of the prevention of deformities by the maintenance of good posture, and finally of muscular reeducation. To achieve these points, both mental and physical rest are important, and if necessary sedatives are required to afford this. During the acute stage, when any movement of the body may cause pain and muscle spasm, the patient should lie on a firm mattress supported by fracture boards with the limbs in the position of rest. Muscle spasm may be relieved by the use of hot foments. In this series four patients had persistent hamstring spasm, which responded gradually but well to the local application of heat. As soon as all pain and spasm have left the muscles, passive and active movements are instituted under the care of a trained physiotherapist.

Abortive Cases.

In the twelve abortive cases, all the patients had typical prodromata with fever, headache and neck and spine rigidity, but developed no paralysis. Two of the younger patients began with convulsions. On examination of the cerebro-spinal fluid, in all cases it was found to contain an increased number of cells and a slightly increased amount of protein. The cerebro-spinal fluid findings were of no help in assessing the progress of these patients.

Treatment.—Treatment in the abortive cases was purely symptomatic. Convalescent serum was not given, as there is no proof that serum given to patients after the onset of symptoms will prevent the development of paralysis.

Encephalitic Paralysis.

The term "polio-encephalitis" should strictly include lesions in any part of the brain or brainstem; such terms as "bulbar", "cerebellar", "hypothalamic" refer to the main features presenting in each case. However, as is usual, the bulbar cases are considered in this article as a separate group.

The clinical features of encephalitis were as follows: fever with severe headache (mainly frontal), meningismus, photophobia, irrationality, and coma of varying degree. Ten of these cases occurred, and in only one was spinal involvement absent. Only one of these patients died, after having been semicomatose for eight weeks; death was probably due to post-encephalitic cerebral degeneration; unfortunately an autopsy was refused in this case. The other nine patients, two of whom were comatose for four to five days, all gradually improved to normal and have remained so to date.

This series of cases provided evidence that the hypothalamus plays an important role in the disease. It has been shown that hypothalamic lesions occur frequently in such cases (Van Rooyen and Rhodes, 1940*a*), and that the lesions are mainly cellular infiltration and œdema in this region. It would be expected that such lesions were to a certain extent reversible, and that they would probably be produced later in the course of the disease than the bulbar paresis, if the pharynx is taken to be the portal of entry of the virus with spread upwards to the hypothalamus via the vagal and glosso-pharyngeal afferents and brainstem. Such a process is in accord with the clinical course in this series of cases.

The onset of irrationality or coma varied from one to nine days, with an average of three days from the initial paresis, and the duration of these manifestations was from two to seven days, with an average of five days. It may be mentioned here that Faber (Murray, 1947) has postulated that the drowsiness and irritability of the prodromal stage are due to invasion of the hypothalamus via the olfactory tracts. In this series of cases it is difficult to invoke a similar process, and the chronological order in which various parts of the nervous system are involved suggests the pharynx as being the more common portal of entry.

There is a remarkable resemblance of the clinical findings in this series to the results of experimental lesions of the hypothalamus (Best and Taylor, 1943): (i) Coma. Stimulation of the posterior lobe produces drowsiness and coma. (ii) Irrationality. When the hypothalamus is released from the control of higher centres a state of sham rage is produced; this state is similar to the emotional instability, talkativeness, and general behaviour of several of these patients. (iii) Disturbance of metabolism. No investigations were made relating to this, but Jangeblut and Resnick (Van Rooyen and Rhodes, 1940*b*) have described abnormal glucose tolerance in monkeys with experimental polio-encephalitis. (iv) Disturbances of autonomic function. (*a*) Gastro-intestinal peristalsis is under the control of the hypothalamus; one patient was irrational and then comatose for two days, and during this period developed pronounced paralytic ileus with fæcal impaction and retention of urine. The ileus recovered in two days, physostigmine (one two-hundredth grain) being given subcutaneously every eight hours. (*b*) Pupillary dilatation results from stimulation of the hypothalamus. This occurred bilaterally in two cases and unilaterally in other cases, and may have been due to involvement of the Edinger-Westphal nucleus, since oculomotor paresis was present in most of these cases. (*c*) With regard to the heat-regulating mechanism, there was no constant feature in the pyrexial reactions of these patients; four of them had a normal temperature during the period concerned. However, all patients sweated profusely at intervals, irrespective of the pyrexial reaction. This is a feature of lesions of the hypothalamus. (*d*) As to vasomotor reactions, the appearance of a rapid pulse, the occurrence of extrasystoles, and sudden halving or doubling of the heart rate are features of experimental lesions of the hypothalamus, and were common in this series. This may have been due to involvement of the circulatory centre in some cases.

Although lesions of the extrapyramidal structures are commonly found (Swan, 1938), there was only one patient in this series with an extrapyramidal syndrome. This patient showed pronounced athetoid arm movements, which subsided after a few days. He also showed gross astereognosis and intention tremor; these may have been due to cerebellar involvement. The numerous cases of nystagmus may also have been due to involvement of the cerebellar system.

Cortical lesions are rare; only one patient in this group showed evidence of a cortical lesion, developing what was probably post-encephalitic gliosis with oculogyric crises, epileptiform seizures and coma.

There was no evidence of an upper motor neuron lesion in the whole series including the spinal group. Cases showing evidence of such a lesion have been mentioned recently in British reports (Murray, 1947).

Treatment.—For the comatose patients, artificial feeding was instituted—by gastric feeding when possible, or if that was impracticable, owing to vomiting, by the intravenous drip method. Restless and irritable patients were not given sedatives if there was any involvement of the respiratory function. Lumbar puncture was used to relieve any excess of cerebro-spinal fluid pressure.

Bulbar Paralysis.

In the fourth group the most important factor is pharyngeal paresis; this leads to impairment of the second stage of deglutition. The nuclei of the cranial nerves controlling the muscles involved are closely related to one another in the region of the medulla and the adjacent fourth ventricle—namely, the dorsal nucleus of the vagus and the *nucleus ambiguus*, with the *tractus solitarius* intervening. The afferent nerves supplying the mucous membrane of the pharynx run to this area. The circulatory centre is in or near the dorsal nucleus of the vagus, and the respiratory centres are scattered in two more or less separate

groups, the lower being under the control of the upper, which lies in the upper part of the pons. If it can be postulated that the portal of entry is via the afferents of the vagus and glosso-pharyngeal nerves from the mucous membrane of the pharynx, all these structures are likely to be affected, and also extension to adjacent structures is likely: below, the cervical cord and accessory nerve; above, the upper cranial nerve nuclei, hypothalamus and respiratory and circulatory centres. Such a process is in accord with the clinical course in these cases.

The physiopathology of bulbar poliomyelitis therefore presents four principal derangements of function, all of which may be present in the one case. These are: (i) dysphagia, (ii) involvement of the respiratory and circulatory centres, (iii) paresis of the respiratory muscles, and (iv) encephalitis or spinal involvement, due to spread of the disease upwards or downwards from the bulb.

In this series 21 bulbar cases occurred—that is, 23·3% of the total. In the 1937–1938 epidemic, in only 8·5% of the total was the bulb involved. Of these 21 patients, 13 suffered from pharyngeal and respiratory and limb paralysis, seven from pharyngeal and limb paralysis, and only one from pharyngeal paralysis alone. Thus all except one patient had spinal involvement as well. Three patients showed evidence of encephalitis, and in two other cases anoxia may have been partly responsible for the cerebral symptoms.

Other forms of paresis occurring in this group were as follows: oculomotor paresis, five cases; ptosis, one case; pupillary changes, two cases; nystagmus, two cases; facial paresis, three cases; hypoglossal paresis, one case.

The results of these bulbar lesions are as follows.

Palatal, Pharyngeal and Laryngeal Paresis.—The patient may be unable to clear mucus and saliva from the throat. Any attempt to swallow may result in the entry into the larynx of fluids or solids. If vomiting occurs, there is a grave danger that the vomitus may be inhaled, and respiratory infection is likely to follow. Many of these patients appear to salivate profusely, and the presence of a gastric tube, through irritation, may increase the accumulation of material in the pharynx.

Involvement of Circulatory and Respiratory Centres.—If the vital circulatory and respiratory centres are involved, the outcome is almost invariably fatal. There is a faint chance that if the patient's airway can be maintained and other difficulties overcome, the centres may recover in time to prevent death.

This point was illustrated well in the case of one patient of this series with severe bulbar involvement and a pronounced encephalitis with an extrapyramidal syndrome. His vital centres were both involved, and respiration ceased for twelve hours; for this length of time he was maintained in a respirator after tracheotomy had been performed, after which irregular gasping respirations began and breathing gradually returned to normal. His circulatory centre was apparently involved to a lesser extent, and it also recovered.

Paresis of the Respiratory Muscles.—Feeble respirations increase the risk that inhaled material may remain in the lungs; bronchial obstruction, atelectasis and bronchopneumonia were common; at autopsy this was confirmed in every case. Posterior collapse and emphysema were common in respirator patients with obstructed airways. Anoxia is inevitable in such cases, and it is probable that the extent of the damage by the virus is increased, and that the recovery of damaged neuronal tissue is prejudiced by anoxia. Irreversible damage may be caused by anoxia alone.

Spread to Other Areas of the Nervous System.—With encephalitic involvement, the irrational or comatose patient frequently inhales material into the larynx, and may not attempt to expel it.

Diagnosis.—The diagnosis was comparatively straightforward. The prodromal stage was in most cases typical, fever and meningism being the commonest manifestations. The earliest definite signs of bulbar involvement were the onset of a nasal voice followed usually within twenty-four hours by dysphagia. In the more severe cases the paralysis then progressed to respiratory paralysis. All except one patient developed spinal involvement, usually slightly later than the bulbar manifestations. No reliance could be placed on the frequently low cell count in the cerebro-spinal fluid; at least six patients had a cell count below 10 per cubic millimetre in the cerebro-spinal fluid, but developed severe paralysis.

Treatment.—Treatment was based on the derangements of function mentioned above. Accumulation of secretion in the pharynx required repeated aspiration. Portable electric suction machines were used, and sometimes it was possible to allow the patient to use the sucker himself. Suction was in most cases performed through the mouth, but in some severe cases continuous suction by means of a rubber tube passed through the nose was found necessary; in such cases it was found that the tube had to be constantly moved about to keep the pharynx clear. At the same time postural drainage, by raising the foot of the bed on high blocks, was utilized to prevent entry of material into the larynx. Atropine was used in some cases to diminish the secretion of saliva, provided that there was no involvement of the circulatory system; but its value was limited. Penicillin and the sulphonamides were used to offset any respiratory infection. The intravenous drip transfusion of saline, glucose solution and plasma was used until the patient was well enough to be fed via a gastric tube. Gastric feeding was necessary for up to three to four weeks, by which time most patients were able to swallow without any spill-over into the larynx. Oxygen was given intranasally to combat anoxia.

In certain cases, tracheotomy was found to be a life-saving measure. The indications for tracheotomy in these cases were as follows: (i) When, in spite of postural drainage, pharyngeal suction and atropine, it was found impossible to prevent pooling of secretions in the pharynx, with consequent spill-over into the larynx causing respiratory distress with cyanosis, râles in the chest, and laryngeal stridor. (ii) When there was any suspicion of anoxia due to spill-over of material into the larynx, or to apposition of paralysed vocal cords in inspiration. (iii) In any case of pharyngeal paresis in which mental symptoms such as irrationality or coma were developing. In such cases, stupor made the patient oblivious of the accumulation of material in the larynx. (iv) In cases in which excitement caused the patient to resist successful pharyngeal aspiration.

In treating these patients it became increasingly clear that early elective tracheotomy, performed before respiratory obstruction and anoxia could occur, was the ideal treatment; low tracheotomy below the thyreoid isthmus, performed under local anæsthesia, was the method chosen in such cases.

Of the 21 patients with bulbar involvement there were eight without respiratory paresis. Only one of these eight patients required tracheotomy for severe respiratory obstruction, and this patient made a rapid recovery.

The remaining 13 patients all had respiratory paralysis. Three of the first of these severely affected patients died before tracheotomy was instituted. Two patients with severe pharyngeal and slight respiratory involvement recovered with postural drainage, suction, and artificial feeding. The other eight patients all had severe pharyngeal and respiratory involvement and were all subjected to tracheotomy. Three of these patients died in spite of tracheotomy, respirator treatment and transfusions, possibly from involvement of the vital centres. The other five patients subjected to tracheotomy all made a good recovery, two requiring respirator treatment and the other three just escaping this. These five patients would almost certainly have died without tracheotomy.

The care of the patient after tracheotomy required good nursing treatment. Continual aspiration of the pharynx and trachea was necessary, with a rubber tube of the correct calibre for the tracheotomy tube. Postural drainage and turning of the patient from side to side to prevent atelectasis were found to be important adjuncts. In most cases it was found necessary to change the tracheotomy

tube daily, and in some cases in which excessive mucopus collected in the tube, twice daily.

The treatment of patients who have undergone tracheotomy and require respirator treatment presented a special problem. It was found that in some patients with long necks the opening of the tracheotomy tube lay outside the respirator collar, and in other patients inside the collar, when it was necessary to provide an airway to the exterior. This was accomplished by means of a small right-angled metal tube fitting into the tracheotomy tube and attached to a length of rubber tubing passing through the collar to the outside. Suction was performed in these cases by removing the right-angled tube, quickly aspirating the secretions, and replacing the tube. Four of these patients were treated thus and three died. The one who survived is now out of the respirator completely and is swallowing normally. It is essential that there be no obstruction to the airway of a respirator patient, as the forceful movement of the lungs against this resistance rapidly produces dangerous emphysema.

The type of respirator used for these cases was of the box type, providing either negative or positive pressure, and it was found that the negative pressure was more effective in maintaining satisfactory oxygenation of the blood.

Prognosis of paresis differs in bulbar and spinal lesions. In the latter varying degrees of recovery occur, but with very few exceptions bulbar paresis is peculiar in that almost 100% recovery occurs within weeks or a few months. This includes oculomotor, abducent, trochlear, facial and hypoglossal paresis.

Finally, it is interesting to review other reports of the use of tracheotomy in these cases. In the 1937–1938 epidemic in South Australia, of the patients admitted to the Metropolitan Infectious Diseases Hospital, Northfield, there were 15 with pharyngeal paresis alone, of whom three died, and eight with respiratory and pharyngeal paresis, all of whom died. No tracheotomies were performed in these cases.

Priest, Boier and Goltz (1947) performed tracheotomy in 75 cases from a series of 180 cases of bulbar poliomyelitis. Of these 29 patients survived and 46 died. In 10 of these cases the tracheotomy was prophylactic. In the remainder it was considered that life was saved by tracheotomy. In Australia one case is reported by McLorinan (1948), with a successful result, and in England a similar successful case by McAlpine *et alii* (1947).

Conclusion.

In selected cases of bulbar poliomyelitis, tracheotomy is a life-saving measure.

Summary.

1. The general features of the whole series of cases of poliomyelitis are presented.
2. The relevant physiopathology is discussed, including the role of the hypothalamus and the sequelæ of the various derangements of function which occur in polio-encephalitis.
3. The treatment of these derangements is discussed with particular reference to the use of tracheotomy in the bulbar form of the disease.

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EXPERIENCE IN THE USE OF ANTICOAGULANTS.[1]

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THIS paper is based on experience gained in the administration of anticoagulants to 270 patients seen in the past thirty months. The main point which the cases have in common is that control of the dosage was in the hands of the writer, either directly or through resident medical officers. The method of controlling the prothrombin content of the blood was one of the modifications of Quick's test; in about half the cases the source of thromboplastin was "Stypven", while in the remainder it was an acetone extract of rabbit brain or human brain. The 270 cases in which anticoagulants have been administered may be summarized as follows:

A. Therapeutic Administration.

i. Surgical cases	54	106 cases
ii. Medical cases	52	

B. Prophylactic Administration—164 cases.

The frequency with which individual drugs were used was as follows:

Dicoumarin alone	134 cases
Ethylidene dicoumarin alone	113 cases
Heparin with one of above	18 cases

In regard to prophylactic administration, some knowledge of the indications for the use of anticoagulants will be assumed, but the major groups of patients to whom they have been administered will be included:

Patients submitted to Cæsarean section ..	104
Women submitted to other pelvic operations	31
Patients submitted to other abdominal operations	19
Repairs of herniæ	10

In nine of these cases there was a history of a thromboembolic complication in a previous operation or childbirth. The large group of women who had Cæsarean section are part of a series currently being treated at the Women's Hospital, Carlton.

Prophylactic Administration.

Considering the prophylactic group as a whole, there are three points that warrant discussion:

1. *The Degree of Reduction of Prothrombin.*—At the beginning of this work, it was thought that in all cases the prothrombin level should be reduced to between 10% and 30% of normal. Workers in the United States had suggested that for prophylactic use a level below 50% was adequate. At about this time it was found that in the Cæsarean section series of patients to whom ethylidene dicoumarin was being administered, it was not easy to maintain levels below 30% in a lot of cases, but that levels below 50% were giving quite satisfactory results. In the past year, in all prophylactic cases the level has been maintained at below 50%, and the results are satisfactory.

2. *The Length of Time for which Anticoagulants Should be Administered.*—The practice has been to keep up a steady dosage of the drug being used until the patient has been fully ambulant for forty-eight hours. When ethylidene dicoumarin is used, there is a return of the prothrombin content to normal in about forty-eight hours. With dicoumarin it may take as long as a week.

3. *The Results of Treatment.*—The results have been an almost unqualified success. In no patient was a major thromboembolic complication of operation seen. One woman who had undergone Cæsarean section for severe toxæmia of pregnancy was given a full course of ethylidene dicoumarin and allowed home after forty-eight hours out of bed. She returned within thirty-six hours with a classical pulmonary infarction, and this responded to heparin and ethylidene dicoumarin, so that she was home again within ten days and has since been well. Two patients developed thromboses in superficial varices of

[1] Read at a meeting of the Victorian Branch of the British Medical Association on June 1, 1949.

the thigh. So far as bleeding was concerned, two patients had sufficient bleeding from their operation wounds to cause comment. One of these had the incision reopened to allow of evacuation of a mass of soft clot, and later had a blood transfusion. One patient developed mild hæmaturia, but this soon stopped and administration of the drug was continued. Apart from these, in no case was there any untoward happening which might be attributed to the use of an anticoagulant.

In nearly all the cases in this group some thrombo-embolic complication of operation was especially likely to occur, so that the complete freedom from any major complication in a person under the control of an anticoagulant is quite striking. The lack of bleeding complications is typical of cases in which proper control of dosage is employed, especially when ethylidene dicoumarin is the drug used.

Therapeutic Administration.

The cases have been divided into those in which the primary condition for which the patients were put to bed was surgical, and those in which a medical condition had been the main cause of illness.

The surgical cases were as follows: (i) venous thrombosis in the calf, 24; (ii) thrombo-phlebitis, 12; (iii) pulmonary embolism or infarction, 10; (iv) femoral vein thrombosis, eight.

The medical cases were as follows: (i) cardiac infarcts, 32; (ii) pulmonary infarcts, 14; (iii) arterial occlusion in limbs, four; (iv) *thrombo-angiitis obliterans*, two.

In this group it has been thought that the best results are seen in cases in which the aim of treatment is to reduce the level of prothrombin to 20% of normal, and to maintain it at about that point for at least two weeks. After that time it appears safe to maintain the level below 50% as in the prophylactic group.

Thrombosis of Veins of the Leg.

The effect of anticoagulants on the pain and discomfort in the group of 32 cases of thrombosis of veins of the leg was such that a very short experience convinces one of their value. Patients with calf vein thromboses appear to do well on dicoumarin alone if treatment is started early, but femoral vein thrombosis constitutes an urgent indication for the use of both heparin—maintained for thirty-six to forty-eight hours—and dicoumarin. The aim should be to start treatment within six to eight hours of the onset, especially in femoral vein cases, and then a perfect result so far as sequelæ of swelling are concerned may be promised. After this period results are less good, although many patients with calf thromboses do well. To follow up the use of anticoagulants with the use of elastic bandages or stockings once the patient is up, is a matter far too often overlooked; but their use can turn a relatively poor result after two weeks into a reasonably good one after six months.

Thrombo-phlebitis in superficial veins, for example, such as one sees in the large veins of the legs after confinement or in varicose veins, is not in many cases very effectively treated by anticoagulants. Some lesions have been seen to disappear with extraordinary rapidity, while others ran an uninterrupted but slow course to recovery. In either case the condition is neither dangerous nor followed by disabling sequelæ, so that the use of anticoagulants is scarcely a matter of much moment in this connexion.

Pulmonary Embolism and Infarction.

It is in pulmonary embolism and infarction that anticoagulants have an action every bit as dramatic as in cases of leg vein thrombosis. Diagnosis is not always easy, largely because we are not yet too familiar with the clinical recognition of the condition. If post-operative embolism is suspected, then heparin should be used without delay. However, in many cases primary thromboses in the lung occur, especially in medical cases, and after operation on patients who have received a blood transfusion; unless urgent symptoms of distress are present, dicoumarin alone is usually adequate. Once again, the drugs are continued until the patient is ambulant or able to take adequate exercise in bed, for example, deep breathing and leg movements.

Cardiac Infarction.

This is not the place for a discussion of the place of anticoagulants in the condition of cardiac infarction, but rather for a discussion of the use of anticoagulants and their dosage in particular. Granted that their use is justified, then the question arises which drug should be used, what should be the level at which prothrombin may be maintained, and for how long should the anticoagulant be used. Briefly, dicoumarin appears to be quite adequate without the addition of heparin, especially in the first few days after the infarct is developed. Its administration should be continued for at least three weeks, and possibly for longer according to many workers. The level of maintenance at which I have aimed is 20% for ten days, and thereafter 50% or less. The aim of treatment is to prevent thrombotic sequelæ of the infarction in organs other than the heart, and this level of maintenance has proved satisfactory. One difficulty that has been encountered is that in most cases of cardiac infarction there is a sharper fall in prothrombin content of the blood than in others in which the same dosage is used. This means that greater care is essential in the control of dosage until the patient's condition is stabilized. Patients who have some degree of peripheral circulatory failure appear to be the most sensitive of all. However, the only hæmorrhagic reaction seen in these cases of cardiac infarction has been epistaxis, although one patient may have had hæmaturia for a few hours.

In this group of cases no patient died with any clot in the cavity of the heart or in any other organ examined *post mortem*. No thromboses were recognized clinically. The fact that only three deaths in 32 cases occurred is merely presented without comment.

Arterial Occlusion in Limbs.

The series of arterial occlusion in limbs is too small for much comment to be made, but the condition is not very common. No surgical measure to remove the embolus was used in any of these cases, and two of the patients died within seventy-two hours of the start of heparin administration. In all four cases relief of pain occurred within a few hours, and swelling diminished appreciably. In the two fatal cases no post-mortem examination was held. In the other two, gangrene of portion of the foot followed blockage of the popliteal artery, while in the other, the limb was saved after apparent blockage of the femoral artery. All cases were among elderly subjects with cardiac insufficiency of varying degree, and three of the patients had auricular fibrillation.

In the two cases of *thrombo-angiitis obliterans* the effect of prolonged use of dicoumarin for about six weeks was tried. Although pain seemed to be eased, the relief was not dramatic, and operations were decided upon in due course. It is doubtful whether anticoagulants play much part in the management of this condition.

Schemes of Dosage.

Schemes of dosage will be outlined briefly only.

1. *Heparin.*—Heparin has been given intravenously only, either by intermittent injection or by continuous drip in saline. Initially 100 milligrammes are given, and thereafter 50 milligrammes every four hours, the dose after midnight (either 2 a.m. or 4 a.m.) being omitted. In no case has the drug been used for more than forty-eight hours, and in most it was used only for five injections over twenty-four hours.

2. *Dicoumarin.*—"Dicumarol" (Abbotts) has been the form of dicoumarin used, and dosage has been 300 milligrammes initially, and then 200 milligrammes on the second day. Thereafter, dosage has been according to the results of daily tests on six days of the week. When the level is 30% or more, 200 milligrammes are given. If the level is below 30%, none is given. It has been found easier to maintain an even level on the dosage of 200 milligrammes per dose than by the use of smaller ones. Rather more than half the cases of cardiac infarction

present an exception to this rule, at least in the first few days.

3. *Ethylidene Dicoumarin.*—Ethylidene dicoumarin is put on the market in tablet form by Nicholas, and its use has been developed by Dr. Paul Fantl, of the Baker Medical Research Institute. Its action in reducing prothrombin is about as speedy as that of dicoumarin, but it possesses the advantage that after its administration has been stopped the rise to normal takes place faster—usually within forty-eight hours. The dose is substantially bigger than that of dicoumarin, and it has been found satisfactory to control the dosage with estimations of the prothrombin time three times a week. This is done on Monday, Wednesday and Friday, and has been found most satisfactory. The initial dose employed has been 500 milligrammes. On the second and third days 300 milligrammes are given on each day. Further dosage depends upon the amount of reduction of the prothrombin content, but for prophylactic use about 70% of patients have been found to require 200 milligrammes each day. When this drug was used, only one severe hæmorrhagic reaction was seen—the case quoted earlier in which a blood transfusion was necessary.

Comment.—It has not been found easy to keep the blood prothrombin level at 20% when the patients have developed a leg vein thrombosis. However, results when the level fell below 50% have been satisfactory. Patients with cardiac infarction require a smaller dose than that outlined above, and usually a dose of not more than 100 milligrammes daily after the first three days is necessary.

Hæmorrhagic Complications.

For the most part hæmorrhagic complications have been insignificant, especially when ethylidine dicoumarin was used. Most of them have occurred in cases in which there was some thrombotic lesion and in which the aim of treatment was to reduce the prothrombin activity to around 20%. The hæmorrhagic episodes that have been noted were as follows: hæmorrhage from the operation site, four; hæmaturia, three; extensive ecchymoses, three; epistaxis, two. In only the one case quoted earlier was hæmorrhage enough to be considered a difficult complication of the use of anticoagulants. In all the others the dosage was continued according to the prothrombin tests, and no further trouble was encountered.

Should any serious complication involving loss of blood arise, it is of paramount importance to realize that the dose of vitamin *K* which is required to correct the prothrombin deficiency is at least 60 milligrammes of a water-soluble analogue given intravenously, and this dose may need to be repeated at intervals of two to six hours, and also to be combined with transfusion of whole blood to restore blood loss.

Discussion.

When a new drug is used, it occasionally happens that only a few patients need to be treated before one is convinced that it is a valuable means of treatment for that one condition. Spectacular examples of such drugs in recent years have been seen in the sulphonamides and in penicillin. In their way, anticoagulants have a specific and striking effect when administered to a patient who has recently developed thrombosis of a vein in the leg or the lung. A small experience of such cases is convincing. However, when one comes to the question of cardiac infarction, the position is not so clear. Irving Wright, in the large group that have been presented from the United States, concludes that the use of anticoagulants is justified, and we who see relatively few numbers in any one person's experience must look more to United States workers for guidance. It is clear that a good case can be made out for the use of anticoagulants, but whether all patients should be given an anticoagulant as a routine measure requires more study. My own experience leads me to wonder whether the elderly person with an impairment of cardiac reserve before the appearance of an infarct will derive such benefit that it is worth while putting him to quite an amount of inconvenience to receive such treatment. For younger patients in whom the infarct is totally unexpected, I believe that no effort should be spared to make anticoagulants available.

When it comes to the choice of an anticoagulant, ethylidene dicoumarin appears to have many advantages over dicoumarin, especially in the field of prophylactic use. In therapeutic use it has not been found easy or possible to maintain prothrombin levels in the region of 20% in many cases, but rather they escape to levels of 40%. However, the results do not seem to have been any the less satisfactory, although experience in this particular sphere is yet too small for one to be certain of this last fact.

Much has been said about the difficulties and the expense of anticoagulant treatment. These, it must be admitted, are considerable. On the other hand, not one of the anticoagulants at present available is anything like ideal, and we may expect better ones to replace them in the future. In the meantime, much of the present work, even if expensive, is far from wasted. Conditions for which anticoagulants are really valuable are becoming defined; lives are being saved and invalidity is being prevented. When more suitable drugs are available, there will be a solid foundation of fundamental knowledge and practical experience which will prevent many errors.

THE USE OF ANTICOAGULANTS.[1]

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THE use of drugs which interfere with blood coagulation for purposes of prophylaxis or treatment of thromboembolism assumes that there is a primary relationship between thrombus formation and the clotting system. Evidence has been furnished that substances inhibiting some part of the coagulation process will prevent thrombus formation in laboratory animals under certain conditions.

It has been demonstrated that glass cannulæ interposed in large arteries would remain patent for as long as twenty-four hours when the coagulation time of the blood was artifically prolonged to fifteen minutes (Murray and Janes, 1940). Thrombi, however, would form in similarly placed cannulæ in animals with a normal whole blood coagulation time.

Although a variety of drugs capable of delaying blood coagulation are known, only a few have found practical application in the prevention and treatment of thrombotic conditions. These are heparin and the compounds of the dicoumarol group. It has to be realized that the properties and activities of the two anticoagulants are entirely different. Heparin is contained within the mast cells of the tissues and is prepared from cattle lung. It is chemically a polysulphuricester of mucoitin and can therefore be classified as a carbohydrate derivative. It is a true anticoagulant and inhibits probably every step in the coagulation system. This is due to its unique properties. Heparin preparations may contain up to 45% sulphuric acid in ester form and carry an exceptionally strong electric charge. These properties indicate that the interference of heparin in blood coagulation is of a physicochemical nature. Whilst heparin acts immediately, both in the circulation and on shed blood, it has a transient effect only. When given orally it is destroyed by enzymes of the alimentary tract and has therefore to be given parenterally, the safest way being intravenous administration. The aim of heparin treatment is to delay the whole blood coagulation time. In the majority of cases this can be achieved by the intravenous injection of 5000 units.

There are variations in response to heparin. It is therefore necessary to control the effect by the estimation of

[1] Read at a meeting of the Victorian Branch of the British Medical Association on June 1, 1949.

[2] The work has been done with the aid of grants from the National Health and Medical Research Council.

whole blood coagulation time. When venous blood is used, a prolongation of the coagulation time from the normal of five minutes to fifteen minutes at 37° C. appears to be desirable. Since the effect of a heparin injection lasts only two to four hours, repeated intravenous injections are necessary and several attempts to overcome this inconvenience have been made. Preparations containing heparin in oily suspension (Bryson and Code, 1944) or in a gelatine base (Loewe, 1947) have been suggested for intramuscular use. Their activity lasts for twenty-four hours. The advantage of heparin medication is quick action. However, any interference with blood coagulation is a risky undertaking, and hæmorrhages following heparin administration have been occasionally reported (Falconer, 1943). The first step in checking a significant hæmorrhage is, of course, to withdraw heparin. Injection of ten millilitres of a 1% protamine sulphate solution will reduce the coagulation time of the blood to normal. A blood transfusion may often be necessary.

The dicoumarol group of drugs display an entirely different action from that of heparin. In contrast, dicoumarols are synthetic compounds. One of them was originally isolated by Campbell and Link (1941) from sweet clover hay which was spoilt during silage. The

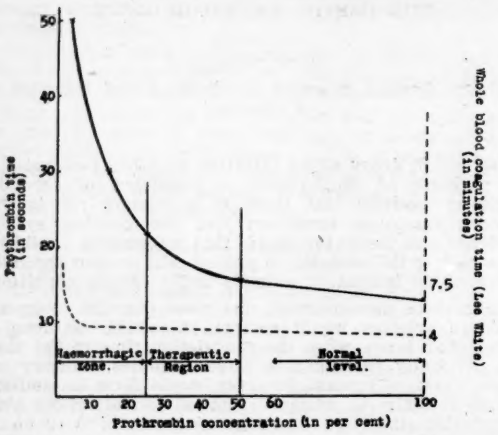

FIGURE I.
Relation between prothrombin concentration and whole blood coagulation time.

dicoumarols have no direct action on blood coagulation—they are not true anticoagulants. They are hepatotoxic substances which, when introduced into the circulation, reduce the ability of the liver to produce prothrombin. They are given orally, and their effect becomes apparent approximately thirty hours after administration. Two compounds in this group have been suggested for practical application. These are dicoumarol and ethylidene-dicoumarol or E.D.C. (Fantl and Nance, 1947). Dicoumarol is more potent than E.D.C. and, further, the recovery from the induced hypoprothrombinæmia is quicker in the case of E.D.C. This gives greater safety in the control of threatening hæmorrhages. Whichever drug of the dicoumarol series is used, great variations in response are observed. Both drug-resistance and hypersensitivity have been found. This fact makes it impossible to give any schedule for treatment. All workers agree and insist that frequent prothrombin estimations on the patient's plasma must be carried out during dicoumarol treatment. Only those techniques of prothrombin estimation which use organ extracts should be employed. The important question is, to what level should the prothrombin content be depressed during dicoumarol treatment? The minimal prolongation of the prothrombin time necessary to obtain a therapeutic effect has not been established. On theoretical grounds one would give sufficient drug to delay whole blood coagulation time. Such a measure would guarantee that no intravenous coagulation could take place, nor could an extension of an existing clot occur. From the accompanying graph (Figure I), however, it is apparent that in the therapeutic zone whole blood coagulation time is not altered. Delay occurs only at prothrombin levels of less than 10%.

Various workers have expressed varying opinions as to the desirable therapeutic prothrombin level during dicoumarol medication. Prior to 1948, according to the Mayo group, intravascular thrombosis rarely occurred when the prothrombin level in the blood was less than 30%, and hæmorrhage was unusual when the percentage of prothrombin was 10 or more (Allen, 1947; Allen *et alii*, 1947).

However, in 1948 E. V. Allen wrote that it was possible that dicoumarol might be administered with satisfactory results if the prothrombin value of the blood was not reduced so much as indicated previously. We are not told the cause of this change in opinion; but presumably it was due to the fact that hypoprothrombinæmia following the dicoumarol medication gave trouble in the form of hæmorrhages. Indeed, it is known from a few reports that fatal hæmorrhages have followed dicoumarol treatment, and the probability is that the number of such published reports inadequately reflects the real incidence of these catastrophes. It should be recalled that dicoumarol was the active agent in spoilt clover hay which produced fatal hæmorrhages in cattle, and it was the economic loss which led to the investigation of the dicoumarol problem. A less potent drug such as E.D.C., which is safer in control, is therefore indicated.

The production of evidence that a less drastic reduction of prothrombin level may produce equally satisfactory results has been claimed by Peters *et alii* (1948). These workers found that prothrombin levels between 40% and

TABLE I.
Results of Treatment of Coronary Thrombosis with Dicoumarol.

Number of Subjects.	Prothrombin Level.	Clinical Embolism.	Mortality Rate.	Hæmorrhages.	Authors.
Treated: 110	40% to 50%	0·9%	10·9%	1%	Peters *et alii* (1948).
Control: 86	100%	15·1%	25·5%	0%	
Treated: 432	10% to 20%	11%	14·9%	12%	Wright, Marple (1948).
Control: 368	100%	25%	24·0%	6%	

50% were adequate for control of the thrombo-embolic conditions in 3000 surgical cases, and also in coronary occlusions. The results obtained by two different groups of workers using dicoumarol in the treatment of coronary thrombosis are compared in Table I.

If we assume that the results in these two groups are due to anticoagulant treatment, then it is apparent that the influence of the drug on the prothrombin level is only one factor in this therapy. Other workers have also held that the mechanism of dicoumarol action is more complex (Gilbert and Nalefsky, 1948).

It does not seem altogether reasonable that the favourable results are due to the anticoagulant action of these drugs alone. Anticoagulant therapy cannot alter the already formed thrombus, whatever effect it may have upon future thrombotic processes. Further, it has been shown that many instances of occlusion are initiated by a subintimal hæmorrhage. According to Wartman (1948), the latter comprise at least 18% of all cases of coronary artery occlusions. Working upon animals and upon the empty heart, Gilbert and Nalefsky were able to show that dicoumarol increased the coronary blood flow.

The favourable effect resulting from the use of anticoagulants seems therefore also to depend on their action as vasodilators.

With regard to venous thrombosis and pulmonary embolism, there is one most desirable goal—namely,

absolute prevention. The magnitude of the problem can be gauged from one example. In order to save six lives from fatal pulmonary embolism, 800 odd patients who had undergone abdominal hysterectomy would have to be treated by anticoagulants (Allen, 1948). This is unfortunately beyond the facilities of most hospitals. It would therefore be desirable to detect the predisposition to venous thrombosis. Thrombosis and embolism follow confinement to bed or generally any condition which is accompanied by stagnation in the venous circulation; for example, it has been reported that sitting in one position for several hours (air-raid shelters, deck chairs) may be followed by a thrombotic attack (Simpson, 1940). An approach toward ambulatory activity during illness or following operation would appear to be a logical prophylactic measure. It is the experience of the majority of observers that muscular exercise soon after surgical interference reduces the incidence of fatal embolism. Muscular activity is known to stimulate the sympathetic system and to activate an enzyme in blood which removes fibrin. It is possible that the process of fibrinolysis is an important factor in the prevention of intravascular coagulation (Fantl and Simon, 1948).

The insidious onset of thrombotic conditions and the unexpected appearance of pulmonary embolism without premonitory symptoms make it desirable to have a reliable biochemical test for the selection of patients requiring treatment. All the proposed tests centre round the assumption that increased coagulability of blood is a predisposing and essential factor in the causation of intravascular thrombosis. The largest number of papers have been devoted to the estimation of whole blood coagulation time. Now, on the surface that appears to be a very simple procedure. One collects either venous or capillary blood and watches for the moment when it sets to a gel. It has been recognized by all students of this phenomenon that it is influenced, not only by chemical factors, but also by physical characteristics, such as temperature and the size and shape of the tube in which the blood is collected—or, in other words, it is a very intricate affair. Of the two techniques, estimation in venous blood is more reliable, because it is possible to control the conditions of the test. Yet capillary blood is used by several workers.

TABLE II.

Influence of Room Temperature on Whole Blood Coagulation Time.
(Capillary Technique.)

Subject.	Prothrombin *per Centum.*	Coagulation Time in Minutes.	
		21° C.	24° C.
J.C.	5	15	9
Mr. C.	100	6	3½
Mrs. C.	100	7	4½

The importance of standardization of this test can be seen from the following example. It concerned the investigation of the hæmorrhagic tendency in a female "bleeder" (J.C.), who suffered from idiopathic hypoprothrombinæmia. Simultaneously with the patient both parents were investigated (Table II). These results indicate clearly that a difference in room temperature of only 3° C. gave for the patient a whole blood coagulation time which would have been considered normal at 24° C. unless a control test on normal blood had been carried out at the same time.

A test which has been suggested by Shapiro (1944) rests on the assumption that greater prothrombin activity is the predisposing factor for thrombus formation. The estimation is carried out in diluted plasma, and a shorter coagulation time is considered to be due to hyperprothrombinæmia. The test as carried out by Shapiro does not consider the fibrinogen concentration in the plasma. However, this is an important factor, because there is a peculiar relationship between fibrinogen concentration and clotting time.

When prothrombin estimations were made on blood from a series of normal donors, which gave similar clotting times in whole plasma, considerable variations were observed when saline-diluted plasma was used, as can be seen from the graph in Figure II.

Fibrinogen estimations indicated that those specimens with a higher fibrinogen concentration had shorter clotting times in diluted plasma. Thus from two plasma specimens having identical prothrombin concentrations, the one with the higher fibrinogen level will indicate an apparent hyperprothrombinæmia, whilst the other with the lower prothrombin level will indicate hypoprothrombinæmia.

Another factor which has been considered of significance in thrombus formation is fibrinogen. From the coagulation scheme it is apparent that fibrin is formed by the action of thrombin on fibrinogen. The mechanism of this reaction is still a matter of dispute. However, it is known that fibrinogen is a large molecule, and that there are intermediates between the start and the end product. The precursors of fibrin have been described by various workers as soluble fibrin (Hammarsten, 1879), profibrin (Apitz, 1939), and fibrinogen B (Lyons, 1945). They are considered by Cummine (1949) a major factor in the onset

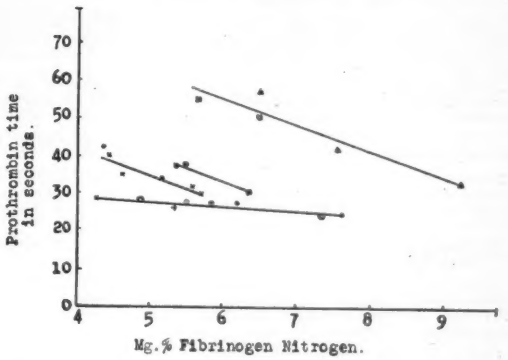

FIGURE II.

Relation between prothrombin time and fibrinogen concentration in 12·5% diluted plasma.

of intravenous thrombosis. Fibrinogen B has been found to occur in 52% of medical and 57% of all surgical patients (Dunn *et alii*, 1949). On the other hand, the incidence of recognizable thrombophlebitis in surgical patients after operation is between 1% and 3% (Homans, 1947). Whilst it seems to be a fact that thrombosis of the lower extremities is found in more than 50% of persons who have died of any cause in the latter half of life (Hunter, 1941), fatal embolism occurs about once in every 1000 operations and may be the cause of 5% of post-operative deaths (McCartney, 1945). From these figures it is apparent that phlebothrombosis may occur more commonly than hitherto suspected. Professor E. R. Trethewie, in Adelaide, supplied me with the following data regarding the occurrence of fibrinogen B. (The test was carried out on the plasma of patients who had been operated on for some condition below the level of the umbilicus.) Of 53 patients in whom fibrinogen B was found to be present, signs of femoral thrombosis developed in five instances *plus* a further four doubtful instances, and pulmonary infarction developed in two instances *plus* a further doubtful instance. On the other hand, in 55 instances in which fibrinogen B was absent, femoral thrombosis developed in one, with a further three in which there was some doubt of the diagnosis, and pulmonary infarction developed in one, and in a further three its occurrence was doubtful. In view of these results, it appears that independent of the absence or the presence of fibrinogen B, thrombosis occurred, which of course lessens the practical value of the test. I have mentioned before that fibrinogen B is an intermediate between native fibrinogen and the fibrin clot. We should then expect that fibrinogen

B, being more closely related to fibrin, would clot more readily than fibrinogen. This should be reflected in the whole blood coagulation time. In other words, in all cases in which a positive response to the fibrinogen B test is obtained the whole blood coagulation time should be shortened. This, however, has not been found to be the case.

From my remarks it should be apparent that a good deal of study has been devoted to the problem of thrombosis and embolism. From the point of view of an experimentalist, however, many more questions have been raised than we are able to answer.

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THE BACKGROUND TO THE ANTICOAGULANTS.[1]

By NOEL M. GUTTERIDGE,

From the Medical Research Foundation, Brisbane.

Thus, many a groan
Heaving, we navigated sad the streight,
For here stood Scylla, while Charybdis there,
With hoarse throat deep absorb'd the briny flood.

—Homer's *Odyssey*, Book XII.

WHEN Ulysses returned from the war in Troy he had to sail through the Straits of Messina. These straits were beset to the right with rocks known as Scylla and on the left a whirlpool—Charybdis. If one considers the rock Scylla as coagulation and the whirlpool Charybdis as fluidity, if we regard the boat as the patient's circulation and Ulysses as the physician who is urging the rowers, first on one side and then on the other, to keep the boat on a straight course, we have a useful concept of the modern attitude towards the maintenance of fluidity and coagulation of the blood.

Just as coagulation is more than a negative fluidity so fluidity is more than a negative coagulation. Macfarlane (1945) best summed up the situation by describing it is a "constantly operating dynamic equilibrium". There are some twenty factors which operate in the body to maintain this dynamic equilibrium. Half of them are coagulation promoters and the other half are fluidity promoters. Most of them oppose each other, but some of them work independently. These factors (*a*) are normally present in the body, but may vary in pathological conditions, or (*b*) can be deliberately introduced into the body, or (*c*) are drugs given for some other condition but which have as their side effect an influence upon this equilibrium. It is convenient, therefore, to discuss first those factors which oppose each other and to consider them together.

Prothrombin and Dicoumarol.

Prothrombin is a coagulation promoter. The precursor of prothrombin is vitamin *K* or 2-methyl-3-phytyl-1-4-naphthoquinone. It has been estimated that a daily intake of one milligramme of vitamin *K* by mouth will supply the body's needs. Vitamin *K* is also synthesized by the bacteria in the intestine. There is one time during life when there are no bacteria in the intestine, and that is at birth. Intestinal antiseptics, such as "sulpha" drugs, will diminish the bacterial production and result in a corresponding reduction in vitamin *K*. The taking of liquid paraffin over long periods interferes with the absorption of many nutrients, including vitamin *K*, and hypoprothrombinæmia has been found to follow as a result. Vitamin *K* deficiency is very rarely seen in patients suffering from severe malnutrition or frank deficiency syndromes. Prothrombin levels in their blood are surprisingly normal.

Vitamin *K* is absorbed into the portal circulation from the intestine, and in the liver is converted to prothrombin. Bile salts are necessary for this absorption. When the absorption of vitamin *K* into the bowel is interfered with, owing to absence of bile salts, as in obstructive jaundice, there is a low level of prothrombin in the blood and hæmorrhages accordingly occur.

Normally there is 0·03% of prothrombin in the blood (35 milligrammes per 100 millilitres). Prothrombin is constantly consumed in the body, possibly in the lungs. It is estimated in plasma by Quick's prothrombin-time method or some modification of that technique. The result is usually expressed as a percentage concentration compared with normal. Unless the thromboplastin used in the test has a consistent activity, the expression of the result in time instead of as an index is apt to be misleading.

[1] Read at a meeting of the Queensland Branch of the British Medical Association on May 6, 1949.

An interesting observation, which provides an explanation for the tendency of tuberculosis patients to suffer from hæmorrhage, was made by Sheely (1941). He found that the prothrombin index is below 59% of normal in 68% of patients suffering from advanced pulmonary tuberculosis.

The fluidity promoter corresponding to prothrombin is dicoumarol. This factor depresses the prothrombin-forming power of the liver. It was discovered by Link *et alii* in spoiled sweet clover. The story of its discovery, isolation and synthesis is one of the most dramatic in the history of medicine.

Dicoumarol is effective when given by mouth. It acts slowly, but its action is prolonged for several days. This slow but prolonged action is both a delight and a horror: a delight because the drug need be administered only every second day, and a horror because there is no simple antidote to an overdosage. Experience with the clinical use of dicoumarol will be discussed later.

Toluidine Blue, and Natural or Injected Heparin.

Toluidine blue and heparin form a pair of antagonistic agents, the toluidine blue being the coagulation promoter because it inactivates the natural heparin which is normally circulating in the body. The studies of Holoubek *et alii* (1949) and Allen *et alii* (1947) showed that titration of the blood, by means of protamine sulphate, of patients with thrombocytopenic purpura showed that an increased heparin-like activity existed and that this could be neutralized *in vitro* by the use of toluidine blue or protamine. It was later found that these substances could be used effectively to arrest the bleeding in patients suffering from purpura associated with a deficiency in thrombocytes. The dye had no effect on the platelet count or the bleeding time.

Personal experience with a patient (H.W.) suffering from panhæmocytopenia demonstrated the dramatic effect of this dye when administered intravenously (three milligrammes per kilogram of body weight).

Allen *et alii* (1947) described a technique for the titration of the natural heparin in the body in which protamine sulphate is used as a neutralizing agent to the heparin. It has long been realized that the formation of petechiæ and ecchymoses does not parallel the platelet count.

We have in toluidine blue an agent which will control the hæmorrhage due to an excess of injected heparin. The need for such an agent does not often arise because of the quick disappearance of heparin from the body in any case. But should that circumstance arise, the injection of five millilitres of 1% solution of toluidine blue intravenously will control the hæmorrhage due to an excess of heparin.

Heparin when injected for therapeutic purposes prevents the formation of thrombin from prothrombin and delays the clumping of platelets. It requires a serum protein factor for its action. It was discovered by Howell and McLean in 1916 and was later purified. It is obtained from liver (as its name implies), but it is also present in all organs of the body.

Heparin has a quick effect on blood coagulation but disappears quickly from the blood (in two to four hours). It can be administered without laboratory control. It can act as an allergin, and the allergic reaction may be fatal if the drug is given intravenously over an interval of more than seven days. This drug is expensive and the complete heparinization of a patient costs three pounds per day.

The effect of drugs on the coagulating tendency of the body can be determined by the heparin tolerance test (de Takats, 1943). In this test the coagulation time is taken. Ten milligrammes of heparin are then injected intravenously and estimates of the coagulation time are made at intervals of ten minutes by the capillary tube method. The usual increase is between 7·0 and 13·5 minutes, with return to normal values in thirty minutes.

Silica Surface and Natural Anticoagulant.

The work of Tocantins (1946) has given an explanation of the long-observed phenomenon that fresh blood drawn into a glass vessel or through glass tubing will clot much more quickly than if the tube has been coated with paraffin. Tocantins believes that there is a natural anticoagulant in the blood, which may be either an antiprothrombin or an antithromboplastin. He has shown that this natural anticoagulant is readily adsorbed on a silica surface. He has further shown that there is an excess of this anticoagulant in hæmophilia to the order of five to eight times. He found that normal plasma requires 320 times and hæmophilic plasma 1000 times as much thromboplastin as plasma adsorbed with 10 milligrammes of asbestos per millilitre for two hours at 20° C. Fantl and Nance (1946, *a*) consider that an excess of this factor can be a cause of "pseudo-hæmophilia" in females. Studies of the effect of asbestos fibre as a local anticoagulant, at the Department of Dentistry in the University of Queensland, have shown that this is a useful local anticoagulant. Theoretically it should be valuable in the control of bleeding from a tooth socket in a hæmophiliac.

Venous Stasis and Muscular Activity.

Another pair of antagonistic factors, venous stasis and muscular activity, have clinical importance in the management of a medical or surgical patient. Venous stasis supplies a site for platelet disintegration by causing damage to the vascular endothelium through anoxæmia. Venous stasis also prevents the normal destruction of the formed fibrin by the fibrinolysin in the blood. On the other hand, fibrinolysin is most active under conditions of muscular activity.

Bed rest, especially in Fowler's position, constricts the femoral vein, where it bends under Poupart's ligament. A study by Wright (1947) of the effect of radio-active sodium (Na_{24}) injected into a patient's leg vein showed that a Geiger counter placed over the groin for sixty seconds was not affected while the patient was in Fowler's position. At the end of that time the patient was instructed to dorsiflex his foot, with the result that a shower of γ rays was recorded in the femoral vein.

The State of the Vascular Endothelium.

Whether the endothelium is intact or damaged has a determining effect upon the balance of the factors, mainly because under conditions of damaged endothelium (associated with trauma or stasis) there is a liberation of thromboplastin.

Ionized and Non-Ionized Calcium.

Ionized calcium is essential for the conversion of prothrombin to thrombin. There is normally available in the body a considerable excess of the calcium necessary for this conversion. In pathological states, such as parathyreoid disturbance, in which there is a great reduction of the serum calcium content, there is never any evidence of a hæmorrhagic state.

In the performance of an exchange transfusion, in which one litre of citrated blood is introduced into the circulation of a heparinized baby and 950 millilitres are withdrawn, there is a severe reduction of the ionized calcium in the circulation. The baby is likely to die of cardiac tetany, but shows no hæmorrhagic tendencies. This hypocalcæmia needs to be prevented by the injection of one millilitre of 10% calcium gluconate solution for every 100 millilitres of citrated blood.

There is, therefore, no justification for the use of a calcium preparation as a routine coagulant, given either by mouth or by injection. *In vitro*, the use of citrate and oxalates for the conversion of calcium into a non-ionized form is everyday practice, but to argue from that to the use of calcium *in vivo* as a coagulant is not justifiable.

We come now to the unpaired factors in this dynamic equilibrium.

Fibrinogen.

Fibrinogen has a long-shaped molecule and represents 3% of the plasma protein. It is rarely deficient in the body, but examples have been reported of both congenital and acquired fibrinogenopenia. Coagulation is affected only when fibrinogen is reduced to one-tenth of its normal concentration in the body. Cummine and Lyons (1947) postulate an altered form of fibrinogen which is called fibrinogen B. This is formed from fibrinogen A when it loses a sulphydryl group. This fibrinogen B combines with thrombin B to form a clot. I have made over 500 examinations of plasma for fibrinogen B according to Lyons's technique and I have found it to provide a useful guide for the presence of the thrombotic state. It is not, however, an absolute indication and must be interpreted in the light of clinical evidence and the study of the coagulation time. The progress of a patient receiving anticoagulant therapy can be usefully studied by a daily fibrinogen B estimation, and it is my practice to continue anticoagulant therapy until the results of fibrinogen B tests have been negative for five consecutive days.

Thromboplastin.

Thromboplastin is present in platelets, tissue juices and damaged vascular endothelium. It is a lipid. It is known as cephalin when brain tissue is the source. The amount of thromboplastin in circulating blood is not very far below that necessary for clotting. Thromboplastin is a combination of two factors: an enzyme, kinase, and a co-factor of lipoidal nature related to lecithin (Leathes and Mellanby, 1939). It has been found that Russell's viper venom will not clot plasma from which lecithin has been removed by extraction with carbon tetrachloride. It has been found that saliva contains kinase, and it is thought that Russell's viper venom is a possible exaggeration of a normal constituent of the saliva. This possibility throws a new light upon the tendency for animals and humans to lick their wounds and thus control hæmorrhage.

Prothrombin Accelerators.

I refer next to a group of coagulants which have been variously described as prothrombin accelerators (Fantl), factor V (Owren) and labile factor (Quick). Fantl and Nance (1946, *b*), who used a purified prothrombin, found that thrombin formation could be appreciably accelerated by a factor present in plasma from which prothrombin had been completely removed. Owren (1947) described factor V and also factor VI. The absence of these factors causes a hæmorrhagic diathesis that he named "parahæmophilia". Quick's (1943) labile factor is not affected by dicoumarol or prothrombin deficiency. It is diminished in blood by storage. It is thermolabile (56° C.) and it is not adsorbed by aluminium hydroxide.

Digitalis.

De Takats *et alii* (1944) showed that heparin tolerance was greatly reduced by digitalis. This drug opposes the action of both heparin and dicoumarol.

Salicylates.

Salicylates act by prolonging the prothrombin time and correspondingly lowering the prothrombin index. Salicylates lower the response to the heparin tolerance test. Prolonged treatment with large doses of salicylate should be accompanied by the administration of vitamin *K*, which acts prophylactically but not curatively on the hæmorrhagic state induced by salicylates. It has been found that one milligramme of vitamin *K* counteracts one gramme of salicylate. Experience at the Department of Dentistry in the University of Queensland has indicated that much of the hæmorrhage taking place after tooth extraction is due to the pre-operative taking of aspirin by the patient to control the pain preceding the operative procedure.

Post-Operative Changes.

Wright (1947) has analysed and listed changes which normally occur in the coagulation factors after an operation. It has been found that the amount of fibrinogen first diminishes and then rises to twice the pre-operative level by the third or fourth day in all patients after operation. This is associated with a rise in the serum calcium content and in the prothrombin index. There is hæmoconcentration and increased viscosity of plasma after an operation, aggravated by a tendency to vomit or a reduced intake of fluids. The most significant change occurs in the number of platelets, which begins to increase on the fifth day and rises to a maximum on the tenth. This increase has been found to be correlated with the severity of the operation. A parallel phenomenon is an increased tendency of the platelets to adhere together. The phenomenon of an increased number of platelets per unit volume of blood is greatest after splenectomy. Dameshek considers that the spleen produces a hormone which controls platelet growth and delivery. After splenectomy this control is lost, with a resultant increase in the platelet count.

Incidence of Pulmonary Thrombosis.

The weight of evidence strongly supports Cummine (1949) in his contention that the general changes in the blood can affect the lung independently of other possible sites of thrombosis. An interesting pathological feature of thrombosis in the lung is that the general effects are out of all proportion to the size of the thrombus. This is explained by the parallel arterial spasm which is induced by the thrombosis in the veins. When thrombosis occurs in the leg the associated arterial spasm can be controlled by the injection of the paravertebral sympathetic ganglia with a local anæsthetic agent.

Fibrinolysin.

Loewe and Hirsch (1947), studying the effect of heparin on traumatized veins in animals, have demonstrated that (i) red cell clots which are not organized and which contain a minute amount of fibrin disappear completely with heparin therapy; (ii) heparin therapy maintains patent adjacent collaterals and tributaries which ordinarily would become involved in the thrombotic occlusive process. These compensatory collaterals often become enlarged to the size of the originally occluded vessel. It has been found that fibrinolysin is most active under conditions of muscular activity. Fibrinolysis is effected by proteolytic enzymes.

Experience with Heparin and Dicoumarol.

I have treated 57 patients with heparin and dicoumarol, either singly or in combination, but mostly the latter. I have found that these drugs are most effective when given early. Their effective application depends upon the detection of early evidence which indicates their use. I have used heparin by intravenous injection only when the condition was grave. In less serious clinical conditions I have found that heparin given by intramuscular injection has been effective. In the combined treatment I have adopted the routine of giving immediately 400 milligrammes of dicoumarol by mouth and 2500 to 5000 units (depending on the clinical condition) of heparin intramuscularly. Three hours later a further 2500 units of heparin are given and six hours after that a further 2500 units (25 milligrammes), with a further dose of similar size at a further interval of twelve hours. Twenty-four hours after the commencement of treatment 200 milligrammes of dicoumarol are given, but none is given on the third day, to enable evidence to accumulate as to the type of reaction to dicoumarol. The prothrombin index is estimated and the fibrinogen B test carried out at the commencement of treatment and on the third, fourth and subsequent days. By the end of the fourth day, with the evidence available, a classification of the type of reaction to dicoumarol is usually possible.

Patients classify themselves into three types: the hyperreactors, the normal reactors and the dicoumarol resisters. A normal reaction is to require a maintenance dose of 100 milligrammes of dicoumarol every second day. The type of reaction to dicoumarol, however, is not constant, and a hyperreactor may require increased dosage of dicoumarol during the second or third week of treatment.

It is an invariable rule that no dicoumarol is given unless the prothrombin index has been estimated on the morning of that day. I use only the 100 milligramme capsules and I do not give a dose unless the index is above 47%. Even with this rule there is no certainty that the patient will not develop hæmaturia; one patient in my series developed hæmaturia at this level, which cleared up without treatment in three days.

Abbott's dicoumarol has been used throughout, except for five patients treated with ethylene dicoumarol—"E.D.C." (Nicholas). It has been found that "E.D.C." was not satisfactory in reducing the prothrombin index. This experience is in keeping with that of Dunn, of Sydney (personal communication). Fantl and Nance (1947) found difficulty in reducing the prothrombin index in some of their patients when using "E.D.C.". Lehmann (1943) found no advantage in the substitution of an ethylene for a methylene bridge in reducing the prothrombin level.

The best statistical survey of the effect of anticoagulant therapy on coronary thrombosis has been provided by Wright *et alii* (1948), who reported the preliminary results of a study initiated by the American Heart Association in 1946. Eight hundred patients were studied—432 treated patients and 368 controls. There were one-third fewer deaths among the treated patients compared with the controls; 24% of the control patients died, whereas only 15% of the treated patients died.

Furthermore, among each 100 patients in the control group, 36 thrombo-embolic complications were diagnosed clinically. Among the treated patients who were receiving the full therapeutic effect of anticoagulant therapy only 6·5 thrombo-embolic complications per 100 patients were noted. The patients were selected for the treatment or the control group on the basis of their entering hospital on an "odd" or an "even" day of the month.

Summary.

1. There is a constantly operating dynamic equilibrium, which maintains the blood in adjustment between fluidity and coagulation. It is the mutual interaction between these factors which enables local clotting to take place when this is normal, or causes abnormal intravascular clotting to take place under abnormal conditions.

2. An understanding of these factors is essential to a rational application of anticoagulant therapy. First the paired factors and later the unpaired factors have been discussed, with particular reference to new developments and clinical application. A technique for administering the anticoagulant drugs has been described and a short account has been given of experience with these agents.

Acknowledgements.

This study has been made possible by the many practitioners who have kindly referred their patients for the type of therapy discussed. Grateful appreciation is expressed of the valuable assistance of Mr. F. Mills, biochemist, and Miss B. Hallam and Miss D. Spottiswoode, serologists.

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TRICHINELLA SPIRALIS: FURTHER SEARCH FOR INFECTIONS OF MAN IN AUSTRALIA.

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IN 1937 the writer examined small pieces of muscle from the diaphragms of 119 cadavers in Australia and found three of them to contain cysts of *Trichinella spiralis*. The amount of tissue averaged about 0·75 gramme; after being cleared in chloro-phenol it was compressed and was then examined under a dissecting microscope. The bodies had been preserved in formalin so that the tissues were unsuitable for digestion.

In many of the recent surveys in the United States and in European countries pepsin or some other enzyme has been used to digest large (25 or 50 grammes) amounts of muscle. The larvæ are found in the sediment, and many light infections are discovered which would be missed in the one or two gramme quantities used for direct microscopic examination.

In view of these findings the Director of the Division of Veterinary Hygiene, Commonwealth Department of Health, suggested that digestion methods should be applied to a sample of the Australian population.

Muscle tissues from human diaphragms have been examined in the past two years by three methods: (i) microscopic examination of a small amount of pressed muscle; (ii) digestion of a larger amount in artificial gastric juice; (iii) feeding of the tissue to rats, which were killed and examined for living larvæ. The results are given below.

Material and Methods.

Ninety-seven diaphragms were collected from the post-mortem rooms of two Sydney hospitals and 105 from the Sydney city morgue; the population investigated would thus include a large proportion of older people. This would increase the percentage of positive findings, as the larvæ can be recognized in the tissues for many years, probably for the lifetime of the infected person.

About three of every four persons examined were Australian born (74%), 12% were British, 6% were New Zealanders, and occasional persons were from European, North American and Asiatic countries.

Technique.

Whole diaphragms were asked for. These were removed by the staffs of the post-mortem rooms and kept in the refrigerator; they were then collected at intervals of two or three days and examined within twenty-four hours. Surplus fat was removed and the muscle put through a mincer. From this minced muscle portions were taken for compression and microscopic examination, for digestion, and for feeding to rats.

Compression.

The apparatus illustrated by Gould (1945) was used to examine an average of two grammes of muscle with a 40× dissecting microscope. Acetic acid (2%) was added to give a clearer picture. This method is particularly useful for detecting infections with calcified cysts.

Digestion.

Twenty-five grammes of muscle were digested in artificial gastric juice (1% pepsin in 0·5% hydrochloric acid) at 37° C. for about twenty-four hours. A mechanical stirrer similar to that of Newman, DeLamater and McNaught (1936) kept the mixture agitated. After being chilled in the refrigerator the digest was sieved through copper gauze to remove fat and coarse particles, and the sediment was examined for larvæ. One hundred specimens were examined in this manner.

Biological Test: Feeding to Rats.

Rats are very susceptible to Trichinella infestations; according to Doerr and Menzi (1933), doses of three larvæ are sufficient to infect nearly every rat. Where the incidence of infection is low, as in Australia, they are useful as a check on other methods, as muscle from a number of subjects can be fed to the one batch of rats. If the incidence of Trichinella is 1% and the tissues of 20 subjects are fed to one batch of rats, then the probability that more than one case will occur in the batch is 0·017. The feeding method takes less time than digestion and would probably be a more delicate test for living larvæ, although we had no opportunity to make a comparison.

The test rats were fed with amounts of muscle which averaged six grammes from each subject. Three weeks after the last feed they were killed and the muscles were examined microscopically and by digestion.

Results.

Table I shows the results of the examination of 202 human diaphragms by three methods.

TABLE I.

Trichinella Spiralis in Human Diaphragms.

Method of Examination and Average Weight of Muscle Used.	Number Examined.	Number of Positive Results.
Compression (2 grammes)	202	1
Digestion (25 grammes)	100	0
Fed to rats (6 grammes)	159	0

The single positive specimen (Number 123) was from a woman, aged eighty-eight years, born in England. The cysts were old, but were still well marked by the heavy calcification of cyst walls and of the enclosed larvæ. Eight grammes of tissue were fed to a rat, but no infection was obtained, and probably all the larvæ had died long before. Seven cysts were found in four grammes of compressed muscle.

In one other case (Number 90), from a male, aged seventy-five years, born in Scotland and resident for thirty years in Australia, one doubtful cyst was found, resembling in size and shape a Trichinella cyst. It was heavily calcified and could not be identified with certainty. No similar object was found in a search of eight grammes of muscle by compression, or in the digest sediment, or in rats.

Discussion.

Three cases of trichinosis were found in 119 cadavers in Australia in a previous survey (Bearup, 1937); in each case the infections were diagnosed from calcified cysts and were evidently of long standing. The method of examination was by microscopic examination of about 0·75 gramme of muscle from the diaphragms of preserved bodies in the disecting rooms.

In the 200 examinations reported here the tissues had not been preserved, so that larger amounts could be used for digestion and for feeding to rats. The failure to find any living larvæ of Trichinella by either method shows that recent infections are rare in this country. The three positive results found in the previous survey and the one reported here were all in immigrants who had probably been infected before coming to Australia.

Earlier reports of trichinosis in pigs in New South Wales were shown to be wrong; the infections were shown to be almost certainly infections with *Stephanurus dentatus* (Bearup, 1937). However, Trichinella must be brought into this country occasionally with imported stock and in rats, and we must expect an odd clinical case in man. New Zealand was thought to be free from the disease, but in 1932 a case occurred in Wellington. Only one member of a family was affected, although they all shared a meal of fried bacon. The diagnosis was proved by the finding of the larvæ in a piece of excised muscle. The origin of the pork could not be traced and 20,000 pigs in the district were examined without the finding of a single positive result (Lynch, 1932).

The higher incidences lately reported from surveys in the United States of America and other countries are probably due to more exact methods of search for the larvæ. In two recent surveys, in which the number of larvæ per gramme of muscle was counted, 86% had counts of less than 11 per gramme. Counts of less than one per gramme were found in 36% (Wright, 1939) and 49% (Gould, 1942) of all positive specimens.

The methods used in the present survey would be as accurate as those used in the two surveys mentioned above, but would still miss some of the infections with dead larvæ and also some of those with low intensities of less than one larva per gramme.

Summary.

An investigation of muscle from the diaphragm of 202 persons in Sydney showed one, possibly two, Trichinella infections. In the one certain case the subject had old and calcified cysts and was a person born outside Australia.

Acknowledgements.

Many persons helped in this survey and their assistance is gratefully acknowledged. These include members of the staffs of the dissecting rooms at the Royal Prince Alfred Hospital, Sydney Hospital, and the Sydney city morgue, who collected the material. Co-workers in the parasitology Department assisted in many ways, and Mr. J. J. Lawrence, also of this School, examined some of the specimens and calculated the probability of positive findings mentioned in the text.

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A LIGATURE-HOLDING BOBBIN WITHOUT MOVING PARTS.

By JOHN DEVINE,
Melbourne.

IN most types of ligature-holding bobbin there are an inner revolving "cotton-reel" on which the silk is wound and an outer metal casing with a hole or slot to allow of egress of thread from inside the case. An appreciable effort is required to draw out the suture material, withdrawal of which causes the inner reel to turn freely. Sometimes "over-running" of the inner reel, when the operator ceases to pull out thread, is a cause of tangling and jamming.

The little annoyances from jamming of spools are such that many surgeons prefer to hold the reels free in their hands and pull off ligature material as required.

In the American magazine *Life* (March 29, 1948, International Edition, pages 38 and 39) mention was made of "spinning reels" for use in fishing. In these there were no moving parts and the thread was peeled off from the end and not as usually from the side of the spindle. So little force was required to pull the thread off the reel that the weight of a cigarette attached to the fishing line at the end of the rod would cause it to run out. The principle of the fishing-line reel was illustrated by showing that usually cotton is wound off its reel from the side with the reel spinning, whereas in the method illustrated it was pulled off over the end, while the reel does not revolve.

FIGURE I.
"Exploded" view of bobbin with short hollow ligature carrier.

It seemed that this principle should be used in making a surgical "bobbin". Such a type of spool would lend itself for use with a hollow ligature-carrier, and even, when used in conjunction with a hollow needle, for suturing.

The bobbin evolved was made of solid chromium-plated brass, and the "ligature-passers" were made of polished stainless steel tubing, which was threaded into the end of the bobbin, so that one bobbin-casing would take any length or type of ligature-carrier or needle. (See Figures I and II.)

For tying at a depth, with the suture material fixed at the ligature-carrier point (as would be done by ear, nose and throat surgeons tying off bleeding points during tonsillectomy), a variation of the bobbin was made in which a "brake" had been incorporated to stop the issue of suture material at will. For "braking", the inner bobbin is pushed

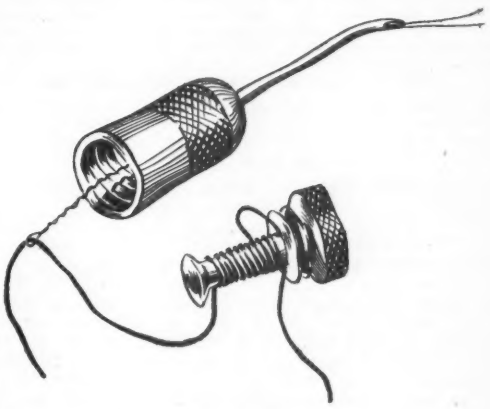

FIGURE II.
Threading the bobbin and ligature carrier (when used) by means of a loop of stainless steel wire.

in from the back so that its end impinges against the ligature material on the edges of the end hole in the outer casing.

For general use the bobbin has been used alone, or with a short (three inches long) slightly curved hollow ligature carrier, which can be "braked" by the thumb while the bobbin is held in the hand (used for under-running vessels — for example, in appendicectomy or gastrectomy).

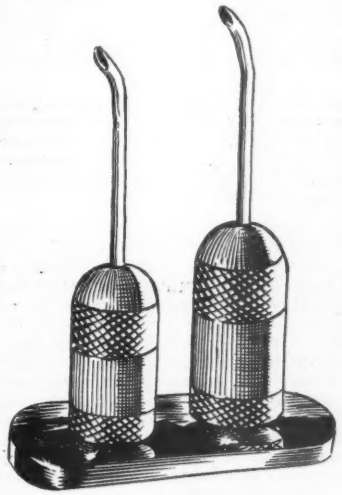

FIGURE III.
A pair of bobbins and short ligature carriers on a rack. When not in use for tying, ligatures can be drawn out of bobbins for threading of needles *et cetera*.

For carrying ligatures to the depths of a wound, an almost straight blunt-pointed carrier of eight inches in length may be used.

For under-running vessels, when ties are to be placed in two places and the vessel cut between (for example, in saphenous-vein ligations and in some techniques for gastrectomy), different coloured suture materials (for example, black and white silk) are wound together and led out by separate holes in the end of a short curved ligature-passer. Thus, when the vessel is under-run the two ligatures are placed at once and the difference in their colours helps to prevent their being crossed.

The bases of the bobbins are bored, and the bobbins can stand on racks on the instrument sister's tray when they are not in use. They are threaded easily and quickly by passing a doubled loop of stainless-steel wire from the point

back through the casing, and putting the ligature material through the loop. (See Figure II.)

In over six months of continuous use, a series of these bobbins has proved most convenient and useful to both the surgeon and the nursing staff. If correctly wound, the suture material issues from the bobbin with almost imperceptible resistance and does not jam.

THE TREATMENT OF PNEUMOCOCCAL MENINGITIS.

By G. A. ROBBIE,
Medical Registrar, Royal Hobart Hospital, Hobart.

IT is proposed to review briefly the treatment of the twelve patients suffering from pneumococcal meningitis who have been admitted to the Royal Hobart Hospital during the past two years. One is unable to contribute anything of great moment to our knowledge of the treatment of this disease; but by following the principles of treatment discussed below we have been satisfied with the results over what admittedly is a very small series of cases.

General Considerations.

Time and Mode of Onset.

All cases, with the exception of Case VII (following a fracture of the skull), occurred in the late winter and spring. This seasonal incidence of the disease has been noted by many, and appears to follow the seasonal incidence of pneumococcal respiratory infections.

The mode of onset varies greatly; in Case III it was as severe septicæmia, in Case VIII the disease certainly began as sinusitis, and in Case II it probably began in the same way as in Case VIII. In Case VII the meningitis began via a fracture of the skull. In Case I the infection may have been an extension from middle-ear disease.

Prior to identification of the organism by microscopic examination of smears and by culture, pneumococcal meningitis as opposed to meningococcal and influenzal meningitis is not easy to diagnose clinically. Probably the devious modes of onset provide the best clinical guide to the offending organism in these cases.

Course of the Disease.

The course of the disease is also variable. When a patient appears to be progressing admirably, a dangerous relapse may occur, presumably owing to rupture of an intracerebral or otherwise loculated collection. Again, a patient whose condition appears hopeless ("dry taps" on lumbar puncture, unconsciousness, high prolonged fever, convulsions *et cetera*) may within a few days make a complete recovery. In no case should one despair. In general the illness is prolonged and trying to treat and hard to prognosticate upon, and relapses during the course of the illness are almost to be expected. Treatment should not be discontinued too soon, since such relapses may be anticipated.

Sequelæ.

Sequelæ, both immediate and remote, were few in this series. One patient (Case VIII) required numerous operations by Mr. H. Trumble. One patient (Case IV) is in the late stages of congestive cardiac failure. There have been no late relapses to date, such as have been described by other writers, months to years after apparent recovery from the original illness. An observation specially applicable to Cases II and III, but also observed to a less extent in two others, was a long period of retrograde amnesia. The memory gradually recovered, but in Case III it took fifteen months; however, recovery is now complete.

Treatment.

Diagnosis.

The diagnosis must be established promptly. If turbid fluid is found on lumbar puncture, penicillin is instilled immediately (and streptomycin as well, if influenzal infection is suspected). Almost always the diagnosis can be made on examination of a direct smear, but a culture is also prepared immediately.

Sulphadiazine.

For an adult, at least four tablets of sulphadiazine every four hours are required to be given orally, or an equivalent dosage of a soluble preparation given intravenously if the patient is vomiting or unconscious. I believe the high dosage can be persisted in almost indefinitely and until the patient appears to be cured. The small danger of sulphadiazine complications is more than offset by the extraordinarily serious nature of the disease. No complications were observed in this series. One patient (Case I), a lad weighing seven stone, was given 610 tablets.

For babies the dosage should not be reduced below one tablet every four hours, at least for the first week to ten days. According to the weight, the dosage for children approaches that for adults, leaning very much to the side of larger doses compared with that for adults.

Penicillin Given Intramuscularly.

High dosage of penicillin, apart from the question of economy, does not appear to be contraindicated. Between 600,000 and 1,000,000 units per day were given to adults. I should prefer higher dosage than this if I had the disease myself. Infants and children were given higher dosage than adults, as calculated by their weight.

Authorities differ about the passage of penicillin through the inflamed meninges; but on general principles, the higher the blood concentration, the more will pass. Other considerations favouring a high dosage are as follows: (i) the need to counter the septicæmic phase which undoubtedly occurs on some occasions; (ii) the need for healing acute endocarditis if this is present; (iii) the need to counter bone infection of ears and sinuses and in skull injuries.

Penicillin Given Intrathecally.

The intrathecal dosage of penicillin chosen for adults was 20,000 units twice a day for four days, and then once a day for at least a further eight days—often much longer. Infants were given half this dosage. No permanent sequelæ arose. However, during the course of this treatment this measure doubtless frequently causes cerebro-spinal fluid pleocytosis, fever and possibly increased irritability. Convulsive seizures or lesser muscle twitchings were not observed as a result of intrathecal therapy with penicillin.

It can be difficult to know when to discontinue intrathecal therapy. In two cases high pleocytosis in the cerebro-spinal fluid and fever promptly cleared when this was done. Since the intrathecal administration of penicillin appears to be a valuable adjunct to therapy, and since no permanent damage appears to result from it, one should persist with this therapy for a considerable period and until one is reasonably certain that all organisms have succumbed. If one encounters a "dry tap" on lumbar puncture, cisternal or ventricular puncture may be undertaken. In my opinion the efficiency of such measures and of through-and-through washes is doubtful. However, they are harmless and not difficult procedures, and one is on occasion driven to these measures in desperation. It has been observed in this series and in several cases of influenzal meningitis that if one encounters a "dry tap" on lumbar puncture even on successive days, spontaneous clearing of the block often occurs with nothing more than the instillation of penicillin by this route when one is sure of having entered the theca.

A thin blood needle is satisfactory for the performance of lumbar puncture in babies.

Collodion and adhesive tape are strongly contraindicated to dress the puncture site; indeed, a sterile sheet to lie on is all that is required.

Heparin was used intrathecally on a few occasions; it did not appear to be efficacious, and at times the withdrawal of blood-stained fluid was the result at subsequent punctures; its use has been discontinued.

Sedation.

Heavy sedation is required, not only to allay restlessness and simplify nursing, but also in an attempt to counter the convulsions which occur not infrequently.

General Supportive Measures.

Serum transfusion was given occasionally, glucose-saline solution on many occasions. Blood transfusion was not required. A high fluid intake, and a urinary output by adults of at least five pints daily were achieved.

Reports of Cases.

The following are brief reports of the twelve cases in the series.

CASE I.—A male patient, aged fifteen years, was admitted to hospital on July 21, 1947, with a history of a discharge from the ear of eight days' duration and of symptoms of meningitis present for one day. He suffered numerous relapses and his life was despaired of many times; but suddenly his condition improved and he went on to complete recovery. He required 47,000,000 units of penicillin intramuscularly, 1,000,000 units of penicillin intrathecally and 310 grammes of sulphadiazine. He was discharged from hospital on September 14, 1947.

CASE II.—A male patient, aged eleven years, was admitted to hospital on September 8, 1947, in a stuporose condition, and with a history of headache and vomiting of three days' duration. He had probably suffered from sinusitis for two weeks prior to this. His condition improved until September 21, when the cerebro-spinal fluid had cleared; but after lumbar puncture on that day the patient collapsed, had violent convulsions and became unconscious. Immediate repetition of lumbar puncture produced grossly turbid cerebro-spinal fluid. On September 29 the patient was still gravely ill and failing to respond. However, from that date onwards he showed steady improvement, apart from several minor relapses. Retrograde amnesia was pronounced after his discharge from hospital on November 20, 1947.

CASE III.—The patient, a female, aged thirty years, was admitted to hospital on September 13, 1947. Her illness had a dramatically sudden onset, with signs of severe septicæmia resembling the collapsed state of fulminating meningococcal septicæmia, but the rash was absent. Her blood pressure was 60 millimetres of mercury, systolic, and 30 millimetres, diastolic. She was deeply unconscious. The cerebro-spinal fluid was crystal clear and contained six cells per cubic millimetre. On September 15 her general condition had slightly improved, but another lumbar puncture produced thick pneumococcal pus. On September 16 no fluid was obtainable by lumbar puncture, and several ventricular punctures were required. On September 22 cerebro-spinal fluid commenced to flow by the lumbar route, and on the same day the patient regained consciousness. Retrograde amnesia was pronounced in this case. The patient was discharged from hospital on November 6, 1947.

CASE IV.—A male patient, aged forty-one years, was admitted to hospital on September 15, 1947. His previous health had been good. He was seriously ill with extensive left lobar pneumonia. His heart appeared to be of normal size on X-ray examination, and no bruits were detected. He was given routine treatment with sulphadiazine and 15,000 units of penicillin every three hours. On September 29 he had failed to respond to treatment and was semi-conscious; pneumococcal meningitis was detected. On this day a bruit was heard at the aortic area, and pneumococcal endocarditis was diagnosted. He responded slowly, but apart from signs of aortic incompetence he was well on his discharge from hospital on November 21. Three months later he first showed evidence of congestive cardiac failure, and radiologically his heart was seen to be rapidly enlarging. At the present time he is in the terminal stages of congestive failure with an enormous *cor bovinum*. He has bequeathed his heart to our museum.

CASE V.—A female patient, aged fifty-four years, was admitted to hospital on November 5, 1947. She had been under treatment for moderately severe hypertension; the meningitis was of sudden onset and clinically severe. She failed to respond to treatment and paralytic ileus developed. She died on November 14. At autopsy it was found that almost complete resolution of the meningeal infection had occurred, and paralytic ileus appeared to have contributed in large part to her death. She also had consolidation of the upper lobe of her right lung.

CASE VI.—A female patient, aged forty years, was admitted to hospital on November 10, 1947. She had been a chronic alcoholic for many years. On her admission to hospital she was delirious and suffering from well-developed meningitis. She rapidly became comatose, and on November 12 she died in *status epilepticus*. At autopsy thick fibrinous purulent exudate was found to be present over the whole surface of the brain and in the ventricles. Gross liver cirrhosis and pronounced fatty degeneration of the heart muscle were present, but the valves were normal.

CASE VII.—A male patient, aged thirty-nine years, was admitted to hospital on April 27, 1948, with a fracture of the skull involving the orbit and the frontal sinus on the left side; the optic nerve had been severed. Consciousness had not been lost. On April 29 his temperature rose to 103·6° F., the patient lost consciousness and meningitis was diagnosed. He remained unconscious for four days. Intensive treatment was required for twenty days. On his discharge from hospital on May 27, 1948, he still complained of a stiff back, but he subsequently made a complete recovery apart from his blind eye.

CASE VIII.—A male patient, aged forty-three years, was admitted to hospital on July 28, 1948, with a history of sinusitis of two weeks' duration, which appeared to clear with treatment. Suddenly he developed symptoms of meningitis. The course was complicated by two violent convulsive seizures followed by aphasia and paresis of the right arm. The aphasia and paresis diminished considerably up to the time of his discharge from hospital on September 1, 1948. However, shortly afterwards osteomyelitis of the frontal bone was detected, and the patient was transferred to Melbourne under the care of Dr. H. Trumble, where an extensive surgical attack was necessary upon the bone and upon both extradural and intracerebral abscesses. He returned to Hobart in April, 1949, and apart from some nominal aphasia he is well.

CASE IX.—A male patient, aged eleven years, was admitted to hospital on August 6, 1948. His disease ran a relatively mild course and rapidly responded to treatment. He was discharged from hospital on August 24.

CASE X.—A male patient, aged nine years, was admitted to hospital on August 10, 1948. His illness ran an uncomplicated and relatively mild course, and he was discharged from hospital on August 30.

CASE XI.—A female patient, aged eight months, was admitted to hospital on August 26, 1948, with a history of pyrexia, vomiting and inability to take her feeds, of four days' duration. She was very slow to respond to treatment, and ventricular puncture was required on several occasions. She had a "swinging" temperature for three weeks. She required 19,000,000 units of penicillin intramuscularly, 340,000 units of penicillin intrathecally and 64 grammes of sulphadiazine. She was discharged from hospital on September 29.

CASE XII.—A male patient, aged fourteen months, was admitted to hospital on November 6, 1948; he had had a high fever initially, and presented the classical signs of meningitis. He responded satisfactorily within four days, but treatment was continued for sixteen days. He was discharged from hospital on November 29.

Summary and Conclusions.

The results of treatment of pneumococcal meningitis in a small series of twelve cases have been gratifying. Two patients died: the habits and general physical status of the patient in Case VI precluded any chance of recovery; the patient in Case V failed to respond clinically despite intensive therapy, and paralytic ileus largely contributed to her death, yet at autopsy the meningeal infection was almost healed. Three cases were relatively mild, but Cases I, II, III, IV, VII, VIII and XI were most severe. One should institute intensive therapy for prolonged periods even in mild cases, and in severe cases one should be prepared to persist with treatment indefinitely or until the patient recovers.

Acknowledgements.

I wish to thank the honorary medical staff of the Royal Hobart Hospital, under whose care these patients were treated. I should also like to thank Dr. W. R. Pitney, clinical pathologist, and Dr. L. W. Knight and Dr. D. E. Anderson, senior resident medical officers, for their invaluable assistance.

Reports of Cases.

HERNIA THROUGH THE FORAMEN OF WINSLOW.

By BARTON VENNER,
Adelaide.

Clinical Record.

A.G., a female patient, aged seventy-six years, was admitted on February 2, 1947, to the Royal Adelaide Hospital in the clinic of Mr. A. Britten-Jones. Being old, non-cooperative and scarcely rational, she was unable to give an adequate history. It was elicited that she had had more or less constant pain in the upper part of her abdomen for about four weeks, and that occasionally her stomach swelled up "like a football". The pain had come on again recently (time indefinite), and for two days she had been retching frequently, vomiting mouthfuls only at a time. At the time of the interview, pain was moderately severe, and aggravated by moving about. The bowels had been opened normally the previous day, but not on the day of her admission to hospital. It could not be elicited whether flatus had been recently passed. Her appetite was poor; she said that she had been passing very little urine, and no dysuria was present.

On her admission to hospital, her temperature was 97° F. and her pulse rate 88 per minute, and respirations numbered twenty-two per minute. She was a pale woman, inclined to be lethargic and non-cooperative. The breath was offensive, and the tongue was dry with a slight fur. No significant abnormality was detected in the heart or lungs; the blood pressure was 110 millimetres of mercury (systolic) and 70 millimetres (diastolic). The abdomen was obviously distended; more localized epigastric distension was present, which produced gurgling under the hand, and was tympanitic on percussion. Severe tenderness was present in the epigastrium and right hypochondrium, with muscle guarding. A hard, irregular, mobile mass was felt in the left iliac fossa and suprapubic region; it was thought to consist of hard fæces. No external herniæ were present. On rectal examination, several small, hard, scybalous masses of fæces were felt, both in the rectum and through its wall.

Soon after the patient's admission to hospital, an enema was given and returned uncoloured, with no flatus, but several hard fæcal nodules. A second enema half an hour later was returned uncoloured, and with no flatus. A third enema yielded three or four more fæcoliths, but still no flatus was passed.

A plain X-ray film of the abdomen was taken. It revealed a large gas bubble in the upper part of the abdomen, apparently in the transverse colon between the splenic flexure and a point several inches distal to the hepatic flexure. (Subsequent events proved this to be a misinterpretation.)

In spite of the general indications of some intestinal obstruction, the apparently stationary condition of the patient and her advanced age led to the adoption of a conservative policy. No further vomiting occurred during the ensuing period of observation; her pulse rate did not reach 90 per minute, and her general condition appeared unchanged. However, the patient died suddenly sixteen hours after her admission to hospital; the sister on duty said that at the time the patient appeared no worse, when she was heard to make a noise resembling vomiting. She was then not dyspnœic, and did not complain of pain, but her pulse rate was rapid and her temperature 95° F. She died within a few minutes. Soon after this the body was examined; no further abnormality could be felt in the abdomen.

Autopsy was performed on February 3. When the abdomen was opened, several ounces of thin, yellow fluid escaped. The stomach was seen to be flattened and pushed forwards by a mass in the lesser sac. The omenta and the mesocolon were discoloured by extravasated blood. The terminal portion of the ileum was traced and seen to enter the foramen of Winslow, together with the hepatic end of the transverse colon, which was stretched taut across the lower border of the stomach. The lesser sac was found to contain the terminal portion of the ileum, the caecum, the appendix and the ascending colon, including the hepatic flexure. These were greatly distended, plum-coloured and friable. The remainder of the small intestine was completely collapsed and flecked with petechial hæmorrhages.

Comment.

Ullman (1924), in his lengthy paper, stated that a pre-operative diagnosis of hernia into the lesser sac of peritoneum had never been made. Hollenberg (1945) reiterates this, and reports a case in which examination after a barium meal and enema together enabled such a diagnosis to be made. The diagnostic points were: (i) evidence of compression of the stomach by an adjacent, gas-filled bowel mass; (ii) evidence of colonic obstruction at a site corresponding to the foramen of Winslow.

Since the first case of hernia into the lesser sac was described by Blandin in 1834 (Haxton, 1944), less than 70 cases of the condition have been reported. In a proportion of these, rupture through the lesser omentum occurred, like that described by Hamilton and Hardy (1946), in their case report and summary. Several causative factors have been described, although none of them appeared to be operative in the case here reported.

A satisfactory diagnosis was not achieved in this case; but the decision against laparotomy was fortunate in view of the findings, for the patient was too frail to have withstood much surgical manipulation. An extensive resection of gut would have been required, and the outcome would almost certainly have been fatal. The condition of the intestine was interesting, the small bowel above the site of herniation being not dilated, as might have been expected. This was probably due to the fact that there was a virtual gastric occlusion, due to the pressure of the contents of the lesser sac on the posterior wall. However, more precise details of the food and fluid intake before the patient's admission to hospital, and of the severity of vomiting, were not elicited.

Diagnosis in this case was somewhat hampered by the patient's lack of insight; but a diagnosis could probably have been made if the condition had been thought of. The presence of localized intestinal distension (both clinical and radiographic) in the upper part of the abdomen, in association with symptoms of obstruction, would probably have justified a diagnosis of hernia into the lesser sac.

Acknowledgement.

My thanks are due to Mr. A. Britten-Jones for permission to publish this case.

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HYDRONEPHROSIS FOLLOWING DIVISION AND LIGATION OF THE URETER.

By V. S. HOWARTH,
Gordon Craig Fellow in Urology, The University of Sydney, and Royal Prince Alfred Hospital, Sydney.

An early, primary, renal atrophy, analogous to the primary atrophy in the liver, pancreas or salivary gland, which follows complete block of their respective ducts, rarely follows complete ureteral obstruction. (Hinman, 1935.)

URETERAL LIGATION has been carried out in many experimental animals and hydronephrosis of a uniform degree is a constant result.

It is still commonly maintained that ligation of the ureter produces primary renal atrophy, and on this account it is felt that the following case history warrants its being placed on record.

In July, 1940, the operation of subtotal hysterectomy for *fibrosis uteri* and a right broad ligament cyst was performed on a female, aged twenty-eight years. The right ureter was displaced and stretched out over the wall

of the cyst and in the course of the dissection was deliberately divided. The proximal and distal ends of the divided ureter were ligated.

An uneventful convalescence ensued, and only slight right-sided abdominal pain, which soon abated, was complained of by the patient.

After this operation the patient was symptomless until she was examined in September, 1947. She then complained of constant pain of four months' duration located in the right costo-vertebral angle.

On cystoscopic examination the bladder was normal, but on catheterization of the right ureter the catheter was held up close to the bladder. The left catheter passed to the renal pelvis and the left retrograde pyelogram showed a normal renal pelvis and kidney shadow.

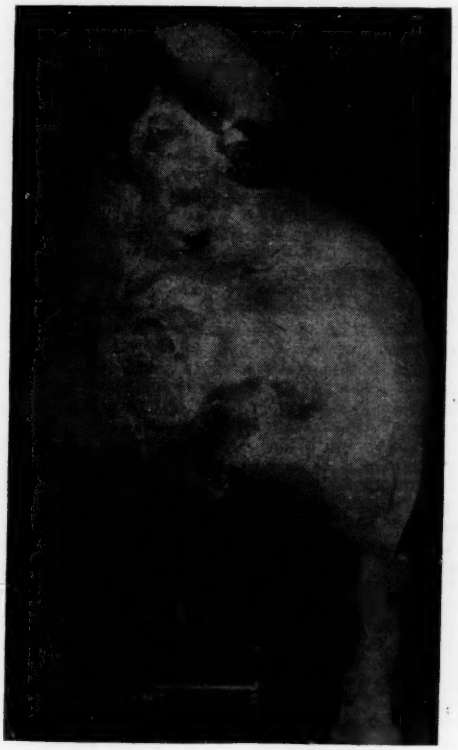

FIGURE I.

Right nephrectomy was performed. Examination of the kidney showed hydronephrosis of the extrarenal and intrarenal type and dilatation of the ureter (Figure I).

Summary.

1. An example of hydronephrosis following division and ligation of the ureter is reported.

2. The long interval is noteworthy between ligation of the ureter and the onset of symptoms, namely, seven years.

Acknowledgements.

I am indebted to Dr. J. W. S. Laidley of the department of urology of the Royal Prince Alfred Hospital, Sydney, for permission to publish this case report, and to the department of medical artistry of the University of Sydney for the photography.

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SJÖGREN'S SYNDROME: REPORT OF A CASE WITH DISCUSSION AS TO CAUSE.

By EVA A. SHIPTON AND ARTHUR D'OMBRAIN,
Sydney.

PATIENTS with a fully developed Sjögren's syndrome are sufficiently rare to justify the reporting of this case.

Clinical Record.

Mrs. M.L., aged fifty-six years, consulted one of us (A.D'O.) on September 11, 1947. She complained of poor vision in artificial light and continuous ocular irritation. Her visual acuity, with a small correction, was six-ninths in the right eye and six-fifths in the left eye. She gave a history of "dry mouth" of fifteen years' duration, with consequent anorexia. On examination of the patient, the slit-lamp microscope revealed a filamentary keratitis. Suspicion of "dry eyes" (Sjögren's syndrome) having been aroused, a litmus paper test was applied, only one millimetre of the paper being wet in five minutes. A mercurochrome test also gave positive results and she was referred to the pathologist for artificial tears.

We are indebted to Dr. K. T. Hughes for the following clinical report.

The patient was first examined on November 17, 1947. The complaint of "dry mouth" was first made fifteen years earlier, whilst she was having dental attention. Her weight then dropped from ten stone to seven stone in five months. At first sucking a lemon caused moistness of her mouth, but for the last eight years she has resorted to barley sugar. For one year her eyelids have felt gritty. The menopause occurred at the age of forty-four years. She has had two healthy children, one male and one female; neither is affected in any way. She has had no past illnesses of note and gives no history of sinus trouble. There has been occasional swelling of the parotid gland on the right side. She gave no history of arthritis, and nothing relevant is revealed in the family history. The patient is edentulous, the teeth having been extracted fourteen years previously on the advice of a specialist, not because of decay.

On examination of the patient, her mouth is dry, and papillæ are still present on the tongue, but the edges are a bright red colour. On pressure over the parotid glands a scanty viscid secretion was obtained from Stenson's duct. The angles of the mouth are dry and slightly cracked. The finger nails are thin and brittle. The hair is dry, but the scalp is well covered and the eyebrows are normal. The skin is dry with a complete absence of sweating and the forearms are pigmented. No abnormality was found in the circulatory or respiratory systems. The urine was normal.

Treatment.

Vitamin *B* was given in tablet form. "Vegemite", with a fluid diet of high caloric value, was prescribed, and two weeks later vitamin *A* and vitamin *D* concentrates were added to the diet and weekly injections of four millilitres of "Campolon" were given. Within four months her weight had increased from seven stone to eight stone four pounds and she had more energy. She said that her eyelids felt less gritty even when she did not use the "artificial tears". Her mouth was still dry, but the tongue had lost the red glazed appearance along the margins, and cracking was no longer present at the angles of the mouth. Eight months after the first examination her weight was eight stone eight pounds, mouth dryness was still present and the right parotid gland secretion was still very viscid. The finger nails were less brittle.

Reports on Laboratory Tests.

On September 12 a blood count gave the following information. The hæmoglobin value was 11 grammes *per centum*; the red blood corpuscles numbered 3,790,000 per cubic millimetre, some small forms being present but no macrocytes; the total and differential leucocyte counts gave normal results. No lymphocytosis and no eosino-

philia was present. A test meal examination revealed a complete absence of secretion of free acid in response to "Bonox". The fasting juice was obtained with difficulty and consisted of only three millilitres of thick mucus. Investigation of the sedimentation rate showed a drop of 70 millimetres at the end of one hour in the Westergren tube.

In August, 1948, the sedimentation rate was still 65 millimetres at the end of one hour, but the blood count showed a slight improvement, the hæmoglobin value being 12·1 grammes *per centum* and the red corpuscles numbering 4,100,000 per cubic millimetre.

The examination of the serum proteins gave the following results: total proteins, 7·0 grammes *per centum*; albumin, 4·19 grammes *per centum;* globulin, 2·35 grammes *per centum*; euglobulin, 0·79 gramme *per centum*; pseudoglobulin, 1·56 grammes *per centum*; fibrinogen, 0·46 gramme *per centum*.

On April 21, 1949, the sedimentation rate was 80 millimetres at the end of one hour, and a blood count showed that the hæmoglobin value was 12·1 grammes *per centum* and the red corpuscles numbered 4,300,000 per cubic millimetre. In spite of the high sedimentation rate the patient volunteered the information that she was better than she had been for years, was taking an interest in life again and had lost the depression which had worried her before treatment.

Discussion.

No satisfactory explanation of the cause of this syndrome has been given. Although the condition bears Sjögren's name, many cases of the disease either wholly or partly developed had been reported previously in the literature.

Sjögren (1943, 1937), considering the frequent occurrence of the ocular symptoms and the hypofunction of the lachrymal glands, with diminution of the salivary secretion, arthritic changes and other general symptoms often found, believed that he was dealing with a general disease and that *keratoconjunctivitis sicca* was only a partial symptom of a larger symptom-complex which depended on infection, and which when fully developed presented in addition, xerostomia, *pharyngitis sicca* and polyarthritis.

Houwer (1927) was the first to point out how frequently patients suffering from filamentary keratitis also have arthritis. In some of his cases the type of arthritis suggested gout, but other reports show great variation in the type of arthritis present.

Gifford, Puntenny and Bellows (1943), reporting on a series of 49 patients, divided them into three groups, as follows: (i) 16 patients showing lachrymal deficiency only —no corneal or associated changes; (ii) 21 patients with fairly severe deficiency with corneal and conjunctival changes and as a rule no associated signs; and (iii) 12 patients with the typical syndrome, seven with arthritis. All but one of the patients were women.

Maclean's (1945) three patients all had chronic arthritis.

The results of laboratory examinations in this syndrome are certainly suggestive of infection. The sedimentation rate is raised in nearly every case. In the case here reported this was extreme, and although the patient's condition improved clinically and the anæmia was relieved, the sedimentation rate remained high. The blood count may be normal; but many patients have hypochromic anæmia, some with an increase in the lymphocyte count and in isolated cases a low degree of eosinophilia (Sjögren, 1937).

The cause of the skin lesions which have been reported in some patients with Sjögren's syndrome (Sheldon, 1938-1939; Weber, 1947) is not known.

Ellman and Parkes Weber (1949) have lately reported a patient with the unusual features of dryness of the bronchial mucosa and pulmonary lesions of uncertain character (probably granulomatous), also arthritis. This patient had normal gastric acidity. They raise the question of the relation of Sjögren's syndrome to Mikulicz's syndrome. This relationship was also discussed previously by Parkes Weber (1945). Ellman and Parkes Weber also draw attention to the fact that the replacement of glandular tissue in Sjögren's syndrome and in Mikulicz's disease by a kind of lymphoid granulomatous tissue is reminiscent of the gradual replacement of thyreoid glandular tissue by lymphadenoid tissue in so-called lymphadenoid goitre.

The relation of Sjögren's syndrome to pernicious anæmia deserves consideration.

Grósz (1936) suggested that the symptom-complex was due to a disease of the blood-forming organs resembling pernicious anæmia, and probably brought about by avitaminosis. It is of interest to note here that Fabian (1937), studying the amount of saliva secreted in gastric carcinoma, pernicious anæmia and gastric achylia, found the quantity reduced in all three; but the reduction was most pronounced in pernicious anæmia—that is, in those patients in whom the gastric mucosa was most severely impaired. The ferments were not affected. Tempka (1937) has shown that saliva collected from a normal person and given orally to a patient with pernicious anæmia could cause a remission of the disease.

Beebe (1936) has reported in detail a patient with xerostomia, hyperchromic anæmia and bilateral parotitis with complete absence of salivary and gastric secretions but normal lachrymal secretion. Improvement followed liver treatment, but the dry mouth and absence of gastric secretion persisted. In other patients observed by him absence of lachrymal secretion, absence of perspiration, loss of vaginal secretion and atrophy of the vaginal mucosa were found. He could not find a satisfactory explanation for the disease, but thought that the generalized atrophy of the salivary, lachrymal and secretory glands of the stomach were in some way related. Beebe was apparently unaware of Sjögren's work as he makes no reference to it; but he was undoubtedly dealing with severe cases of this syndrome.

The first patient reported by Lutman and Favata (1946) had a sister with pernicious anæmia, and their mother had severe arthritis.

Dry skin is frequently seen in pernicious anæmia, and absence of sweating is a feature of Sjögren's syndrome (Sjögren, 1943). In early cases of pernicious anæmia dry mouth is at times the only symptom.

Lisch (1937), in a long, excellent paper, has shown that Sjögren's syndrome is hereditary; in one family deficient lachrymation was present in three generations. Not all the affected members were females.

Coverdale (1948) reports a man and his daughter, both of whom were affected, and also a woman and her daughter. Congenital cases do occur, Hamilton (1938) having seen the condition in a male child aged eleven months.

Some authorities believe that avitaminosis is the cause of the syndrome. This belief is based largely on Stahel's report (1938) of a typical severe case in a female patient, aged sixty-four years, who, amongst all the other symptoms including arthritis, had urethritis and *vulvo-vaginitis sicca*; this patient responded well to vitamin *A*. Here again the analogy to pernicious anæmia is present, as recent research suggests lack of vitamin B_{12} as the cause of this disease. There is, however, little scientific support for vitamin *A* deficiency as a cause of Sjögren's syndrome, as careful studies have failed to show any deficiency of this vitamin in the blood in the majority of cases examined, although many of the symptoms believed due to lack of vitamin *A* resemble those found in Sjögren's syndrome.

Clinically prominent in the second case reported by Lutman and Favata was the implication of a deficiency of the vitamin *B* complex; but treatment with yeast, vitamin *B* complex and riboflavin was unsuccessful.

Saphir (1940) reported a patient with severe xerostomia who responded to nicotinic acid, but not to yeast or thiamine chloride.

When the syndrome is fully developed, with absence of gastric secretion and probably absence or reduction in amount of other digestive juices, it is highly probable that deficient absorption of vitamins and other substances is present.

One of Spector's patients was benefited by pancreatin (Bruce, 1941).

The endocrine theory has had much support, as it is an undoubted fact that the majority of cases occur in females after the menopause; however, cases have been reported in males and in young women and, as has been

noted, congenital examples do occur. Treatment of these patients presents many difficulties. If one bears in mind the precept "*primum non nocere*", the condition makes the patient so miserable and depressed that if one treatment does not succeed another may help, and almost anything is worth trying. Liver, given both orally because of the effect on gastric secretion (Kim and Ivy, 1933) and by injection, may be tried even if achlorhydria is not present. Acid given by mouth, if the mouth is not too sore, pancreatin, and even folic acid may all help the patient to absorb food. Cases of associated hypoglycæmia are on record (Parkes Weber, 1947), and this should be remembered when one is planning the diet. Yeast, vitamin *A* and the vitamin *B* complex may all help. If achlorhydria is present, vitamin *C* in large doses may help.

A wider recognition of this syndrome with thorough clinical and laboratory examination of patients and a careful record of the effects of treatment will help to find the cause of the syndrome.

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BACTERIUM FÆCALIS ALKALIGENES SEPTICÆMIA: REPORT OF A CASE.

By GEORGE HALL and JOHN GARVAN,

Sydney.

PETRUSCHKY in 1896 first isolated from human fæces and stale beer an organism which he called *Bacterium fæcalis alkaligenes*.

The *Alkaligenes fæcalis* group of organisms, probably intermediate from a morphological and cultural point of view between the colon and Brucella groups, are generally regarded as being non-pathogenic. However, *Bacterium fæcalis alkaligenes* has been isolated in association with many diseases of the human subject. It is doubtful if it was the causative organism in some cases, but in a large proportion of them there are adequate grounds to consider it the ætiological pathogen.

Pathogenicity of Bacterium Fæcalis Alkaligenes.

It seems well established that *Bacterium fæcalis alkaligenes* can be pathogenic. Spray and Hawk (1934) isolated it in a case of meningitis secondary to *otitis media*. It is reported as the cause of meningitis following craniotomy by Gatewood (1931) and in association with meningocele by Mason (1934). Anderson (1933) obtained the organism from the blood of a patient suffering from rheumatic polyarthritis, and McIntyre (1936) grew the organism from the blood of a jaundiced patient with acute infective hepatitis. In infection of the genito-urinary tract it has been found in the urine by Stuart, Thomson and Prikorian (1934) and by Ahad (1942).

Perhaps the most interesting series of cases reported include those in which this organism has been isolated from the blood during pyrexial states resembling typhoid fever. Voorhies and Wilen (1942) reported a case with a febrile course lasting for four months with a multiplicity of symptoms, the most striking of which was shifting polyarthritis. Of interest also in this case was the development of symptoms and laboratory evidence of meningitis. The patient exhibited a mild typhoid-like state, and all symptoms subsided after the administration of sulphadiazine. Hazen and Mortillaro (1936) report the association of the organism with an enteric-like fever.

Chaudhuri (1944), Wasti (1945) and Raeburn (1944) report several cases in which the organism was obtained from the blood-stream during pyrexias of varying duration. There is no doubt that the organism has been isolated from the blood in numerous cases of pyrexia, but it is usually discarded as being a contaminant. Hirst reports that of 133 positive results from blood culture *Bacterium fæcalis alkaligenes* was found in 23 (18·7%). Its mode of entry into the blood-stream is unknown.

Clinical Record.

The patient, a male, aged thirty-eight years, was admitted to an Australian general hospital on January 14, 1946. He complained of generalized aches and pains, malaise, fever, general weakness and loss of appetite. The duration of symptoms was three weeks. He stated that he had felt much worse in the last twelve hours before admission, and during this period his testicles had become painful and swollen and joint pains had become troublesome. There were no symptoms referable to other systems. The patient was a repatriated prisoner of war (from Malaya) and had had between 20 and 30 attacks of malaria.

Examination on admission to hospital revealed the patient to be feverish (his temperature was 101° F.), ill, but not prostrated. The testicles were both swollen and tender. The knee, ankle and shoulder joints were painful on movement, but there were no swellings of the joints. The lower margin of the liver was palpable (one finger's breadth below the right costal margin) and tender. The blood pressure was 120 millimetres of mercury (systolic) and 70 millimetres (diastolic). The urine was apparently normal.

There were exacerbations and remissions of the testicular swelling and tenderness for the first two weeks of the illness. Abdominal distension was troublesome throughout the illness, and hepatomegaly increased, the lower margin of the liver being four fingers' breadth below the right costal margin on February 26, 1946. Joint pains in upper and lower extremities also persisted throughout the illness.

On March 27, 1946, seventy-two days after admission to hospital, the patient became unconscious. Meningismus was noted, and two days later the patient developed convulsive movements of the right side of the face and right arm. These movements were followed by generalized convulsions.

On March 30, seventy-five days after admission to hospital, the patient died in coma.

The temperature throughout the illness was of the high remittent type varying from 101° F. to 104° F.

Treatment included courses of sulphathiazole (40 grammes), sulphadiazine (45 grammes) and sulphamerazine (30 grammes). Penicillin was given throughout the illness, the total administered being 9,390,000 units. Trial courses of emetine and carbarsone were also given with no therapeutic response. Two transfusions of citrated blood each of one litre were adminis-

tered. Efforts made to obtain supplies of streptomycin for treatment of this patient failed. At this time (early in 1946) the drug had not been long in clinical use and was in very short supply.

Laboratory Investigations.

On the patient's admission to hospital a blood count revealed the following information. The red cells numbered 4,000,000 per cubic millimetre, the hæmoglobin value was 9·28 grammes *per centum*, and the leucocytes numbered 17,800 per cubic millimetre, of which 94% were neutrophile cells. The leucocyte count rose progressively to 37,000 per cubic millimetre, of which 90% were neutrophile cells; this last count was made a few days before death. There was a persistent hypochromic anæmia.

Blood Culture.

Blood culture was performed weekly after the patient's admission to hospital. On the third occasion the organism was isolated, but several attempts subsequent to this failed to produce any growth. Subculture of the third blood culture from glucose broth on to blood agar grew a Gram-negative motile bacillus, which was slightly larger and longer than the ordinary coliform organism. The colonies were opaque and corresponded in characteristics to those given by Topley and Wilson. There was no fermentation of glucose, lactose, dulcite, saccharose, mannite or maltose. The organism did not produce indole and rendered litmus milk alkaline. From these findings it was classified as *Bacterium fæcalis alkaligenes*.

Repeated examination of the blood failed to reveal malarial parasites. Cerebro-spinal fluid, obtained by lumbar puncture when there was clinical evidence of meningitis, contained 15 lymphocytes per cubic millimetre, and had a protein content of 200 milligrammes *per centum*, and a chloride content of 610 milligrammes *per centum*. Culture on to blood agar produced no growth. Microscopic examination of the urine on several occasions revealed no abnormality, and culture produced no growth. The fæces were repeatedly investigated, but no non-lactose-fermenting organisms could be isolated, nor were cysts or amœbæ seen on microscopic examination.

The patient's serum showed no significant agglutinating titre against suspensions of the Salmonella group of organisms or *Brucella abortus*. The Weil-Felix reaction was absent. There was no agglutination by the serum of a suspension of the organism in the smooth phase isolated by blood culture.

Post-Mortem Findings.

The autopsy was performed sixteen hours after death. There was great wasting of the subcutaneous tissues. The significant findings were fibrinous pleurisy of the left lung and a liver slightly smaller than normal, congested and friable. Microscopic examination of sections of the liver showed extensive cloudy swelling. The kidneys contained irregular subcapsular yellowish nodules which varied in size from a few millimetres to a centimetre in diameter and in section were wedge-shaped. Microscopic examination of these nodules showed the changes seen in a pyæmic infarct of the kidney. There was no evidence of suppuration in the nodules.

The meninges and the brain contained no microscopic evidence of inflammation, except for a small nodule in the region of the right thalamus, which microscopically was found to be a small abscess without suppuration.

Comment.

We believe this case to be of clinical interest and importance because of the prolonged febrile typhoid-like illness (the duration was more than three months) associated with a striking multiplicity of symptoms. In cases reported in the literature the duration of the septicæmia varied from two weeks to two months.

The most striking symptoms in this case were the multiple joint pains, the testicular pain and swelling, and the meningeal reaction in the final stages of the illness. These symptoms have been reported previously by Anderson (1933), Voorhies and Wilen (1942) and others, in association with infection with *Bacterium fæcalis alkaligenes*. The case reported by Voorhies and Wilen (1942) most closely resembled this case, but it differed in the rapid response to treatment with sulphadiazine.

We believe this to be the first case of *Bacterium fæcalis alkaligenes* septicæmia reported in Australia.

We should like particularly to draw attention to the fact that *Bacterium fæcalis alkaligenes* is not infrequently grown from the blood of patients with prolonged pyrexia (18·7% in Hirst's series of 133 positive results from blood culture) but is usually discarded as a contaminant. It seems well established now that the organism can be pathogenic and it may well be the ætiological factor in many of these cases. The recognition of this fact seems to be of particular importance, as one of us (J.G.) has recently encountered another case, clinically similar to the one reported above, in which blood culture produced a growth of *Bacterium fæcalis alkaligenes* and there was a dramatic response to treatment with streptomycin.

Summary.

1. A fatal case of bacterisemia due to a *Bacterium alkaligenes fæcalis* type of organism is reported. The febrile illness lasted for more than three months, the most interesting symptoms being shifting polyarthritis, orchitis and meningitis.

2. A report of the post-mortem findings is given and the literature is reviewed.

3. It is emphasized that *Bacterium fæcalis alkaligenes* is not infrequently grown from the blood of patients with a prolonged febrile illness but is often discarded as a contaminant.

4. It is pointed out that the organism can be pathogenic and may be the ætiological factor in many of these cases.

5. In a recent case dramatic response followed treatment with streptomycin.

Acknowledgement.

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Reviews.

A TEXT-BOOK OF CLINICAL PATHOLOGY.

THE third edition of Dr. Francis P. Parker's "Textbook of Clinical Pathology" appears eight years after the second.[1] It is a laboratory manual, to which eight authors, nearly all of professional status, have contributed sections in their own specialty. The arrangement of the chapters is along clinical lines, and a trifle strange to British eyes not familiar with the earlier editions.

The first section on examination and diseases of the blood is followed by others on blood chemistry, assay of vitamins and hormones; then come tests of liver function; these are followed by examination of sputum, gastric contents, urine, fæces and cerebro-spinal fluid. A chapter on

[1] "A Textbook of Clinical Pathology", edited by Francis P. Parker, M.D.; Third Edition; 1948. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" × 6", pp. 1044, with illustrations. Price: 67s. 6d.

immunological tests in general and a consideration of transudates and exudates, skin and mucous membranes precedes the final sections on direct diagnosis of venereal lesions and the serological tests for syphilis.

The practical approach is very noticeable, each chapter including remarks on interpretation of tests, fallacies dependent on factors without and within the laboratory and the collection of specimens, and sources of supply in the United States of many reliable reagents are stated for the information of those people blessed with dollars.

It is interesting to observe the differences in illustrations in the various sections, in all likelihood expressing the attitudes to such things of the authors. The graphs of composite and separate analyses of leucocytes, and of marrow composition during age periods, are informative and useful. The technical discussion on blood groups is good, and in relation to the Rh factor the various tests have been followed by brief explanations only, and the differences of nomenclature and significance are not extremely clear. In this country there is a general opinion that the subgrouping of the Rh factor is best left in the hands of experts, since the reagents for the potency testing of sera can hardly be included in the equipment of a routine laboratory, and Dr. Davidsohn might well have voiced a warning in this matter.

The sections on blood chemistry are clear and detailed and the discussion of tests for liver function particularly sane and balanced, but the relative significance attached to various tests of renal function is different from standards held in this country.

The discussion of immunological tests chiefly in the indirect diagnosis of infectious disease includes numbers of tests not practicable in this country. Brucellosis and tularæmia are not the problems that they are in the United States, and clinical pathologists have not developed techniques to such an extent. In some sections, references to complement-fixation tests and antigen preparation assume the presence of equipment and personnel not likely to be found in many Australian laboratories.

The chapters on the serological diagnosis of syphilis go into great detail and set out modifications of methods, and deal with reasons for failures in technique which are very useful.

Detailed reading of the text will reveal many small points on which the experienced laboratory worker will argue fiercely, such as the statement that a negative response to the Schick test indicates that the subject possesses "sufficient natural antitoxin to protect against diphtheria".

The book is printed on beautiful paper and in clear text; the author died while it was still in page proof, and there are many small errors of spelling attributable to this fact. It is a book which will interest and stimulate the senior man; but is likely to be a mixed blessing to the beginner, because so many debatable and partially proven techniques are included amongst those which are established as routine procedures. The bibliography is good. The index is not complete.

FORENSIC MEDICINE.

SYDNEY SMITH'S well-known text-book of "Forensic Medicine" is now in its ninth edition.[1] In the publication of this new edition he has had the cooperation of Dr. F. S. Fiddes, who is introduced to the reader as the joint editor. The authors have embarked on no major rearrangement in this edition apart from a general revision of the text and replacement of some of the illustrations. New information has been included on *rigor mortis*, on the Rhesus factor, and on the value of British anti-Lewisite in the treatment of poisoning by some of the heavy metals. In the section on *rigor mortis* reference is made to the interesting work of Szent-Györgyi on muscular contraction.

In the general post-mortem examination conducted in medico-legal cases, when death has been due to violence, inspection of the body a second time may be helpful, especially if the autopsy has been conducted a short time after death. It may be noted on many occasions that twelve to twenty-four hours after death, marks on the body, especially bruises, are much more apparent than they are shortly after death.

In the section dealing with sudden death from inhibition the statement is made that in these cases the autopsy does not assist in the diagnosis of the cause of death. This statement we feel provides criminal barristers with more than they are entitled to, and this section has been used in court against medical experts. It is true that death from "vagal inhibition" is not an organic diagnosis, but nevertheless it is strange to say that the autopsy does not assist in the determination of the cause of death. By a process of elimination at the autopsy of any other cause of death, with the history usually available, together with the autopsy findings, for example, injury to the vagina or uterus in attempts to procure abortion, the significance of the autopsy in the making of the diagnosis cannot be over-emphasized.

In the section dealing with the examination of "Hairs and other Fibres" there is a chart dealing with cuticular scale patterns. It appears from the table on page 101 that the type of scale pattern shown in column four is specific for the black-faced sheep. We have seen this particular scale pattern also in the dog. Where the healing of wounds is discussed, there is greater scope for more precision in estimating how long before death wounds have been inflicted. Microscopic changes may be observed, as the authors indicate, long before a twelve-hour period elapses. Stuffing of capillaries with leucocytes and their emigration into the surrounding tissues may be observed in less than one hour.

The discussion on ethyl-alcohol is a good one, and as the examination of intoxicated individuals and subsequent court proceedings are one of the bugbears of medical practitioners, the information given will prove of the greatest use. Post-mortem examination of the blood for its alcoholic content, or its absence, may also be of much value. The importance of securing a sample of blood for this purpose is indicated in the chapter on the autopsy.

This book needs no praise. It has taken its place as a standard work on "Forensic Medicine" and is widely used by both the medical and legal professions. Those who are constantly engaged in this type of work fully appreciate the wealth of valuable material that it contains.

[1] "Forensic Medicine: A Textbook for Students and Practitioners", by Sir Sydney Smith, C.B.E., M.D. (Edinburgh), F.R.C.P. (Edinburgh), D.P.H., and Frederick Smith Fiddes, O.B.E., M.D.; Ninth Edition; 1949. London: J. and A. Churchill, Limited. 8½" × 5½", pp. 676, with 173 illustrations. Price: 30s.

THE SCOTSMAN'S FOOD.

A SMALL, unpretentious volume, "The Scotsman's Food", bound in thin cardboard, has been written by A. H. Kitchin and R. Passmore, with an introduction by Professor F. A. E. Crew, ostensibly as a defence of food rationing with special application to Great Britain north of the Tweed.[1] But the book is much more than this; it is an admirable if abbreviated history of nutrition with far more than local interest, carrying the reader on to the very modern era of government planning. After an elementary presentation of the main basic types of food constituents there is an excellent summary of the factors affecting food production and of those affecting food consumption. There are indeed some grim chapters in the story of nutrition, not confined by any means to Scotland. Bad seasons leading to famine and pestilence and consequent appallingly high death rate were once all too common. Conditions could have been better had the farmer been more responsive to suggested improvements in agriculture; in some cases his stupid and obstinate antagonism, for example, to the introduction of the potato, cannot be pardoned. Starvation and disease were sent as divine punishments for sin and their inevitability dare not be questioned. The authors quote from "a not unsympathetic historian" as follows: "Piety dignified dirt and consecrated laziness. People believed that disease was due to the hand of God instead of want of using their own hands; that dearth was due to Providence rather than their own improvidence." Yet the authors admit that the diet of rural Scotland in the past, though monotonous and precarious in its supply, was yet at its best of high physiological quality. "On the whole, however, the simple but nutritious diet of rural Scotland built up a people of excellent physique." It was in the much lauded industrial period of the nineteenth century of which the subjects of Queen Victoria were so proud that malnutrition assumed its most horrible phases. It was then that Glasgow gained the evil repute of having rickets endemic. "Here we wish to emphasise that the apparent industrial prosperity of the second half of the nineteenth century, as represented by dividends of twenty-five per cent., was biologically a sham."

The latter part of the book is a description and defence of food rationing—"a dietary uniformity has been imposed in a degree perhaps unique in history. Geographically but

[1] "The Scotsman's Food: An Historical Introduction to Modern Food Administration", by A. H. Kitchin, M.B., and R. Passmore, M.A., D.M., F.R.C.S., with a foreword by Professor F. A. E. Crew, F.R.S.; 1949. Edinburgh: E. and S. Livingstone, Limited. 7¼" × 4¾", pp. 96, with seven illustrations. Price: 3s. 6d.

few local dietary habits and customs remain. Social distinctions have also been much reduced. There has been a levelling both upward and downward. The underfed tenth disappeared early in the war years". The authors quite candidly admit that this condition has been bought at a price. "It is equally certain that it has not been achieved without a reduction in both the quantity and quality of the food consumed by large numbers of the hitherto economically favoured. There has been a restricted consumption of many desirable and health-giving foods, the abandonment of many healthy food habits, ingrained in families for centuries, and the loss of innumerable pleasant social customs." Then come descriptions of straight rationing, the points system, welfare foods for the processes of growth and reproduction such as free cod-liver oil to all expectant mothers and children under the age of five, school meals, industrial canteens, food subsidies, the black market, which is shown to be a mere shadow of what exists in the European continent, fortification of foods, for example the addition of vitamins to margarine, and allotments for home vegetable growing. The usual attack on white bread has not been forgotten. In the final summary it is admitted that the data are not available to make any specific statement regarding the present health of adults, but "probably never before in history and certainly not within living memory have the children of Scotland been so healthy". Though there are descriptions of certain procedures and regulations unknown in Australia, this fact does not prevent this brochure from possessing a high value for readers in this Commonwealth. It deserves wide circulation, not only amongst university students to whom it was primarily addressed, but amongst the people as a whole.

Perhaps it is the calm detachment of the authors which has led to a certain disappointment that a truly Scotch touch is not discernible. There is no laudation of oatmeal; in fact the rather condensed statement about cereals on page 7 might make many readers believe that in oatmeal "only traces of fat are present". Oatmeal in Australia contains 8% to 10% oil and there is no reason to expect a lower concentration in Great Britain. Scotland is a "land of cakes", and its fruit cake and shortbread still retain their pre-eminence. Where is there a better conserve than Dundee marmalade? Then, sad to relate, there is not a single mention of the haggis—"great chieftain o' the puddin' race". Robert Burns ridiculed the spindle shanks, weak fists and food snippets of the Frenchman—"Poor devil! see him owre his trash"—and then passed to the Scot:

> "But mark the Rustic haggis fed,
> The trembling earth resounds his tread."

A more serious criticism is that the high place of fish in the Scots diet, and indeed in the British diet in general, has not been emphasized as it might. An Australian dietitian who recently visited the Old Country has declared "much of England has reared itself on fish and chips". Scotland has a relatively large coast line and the fishing industry is organized in a manner unknown in Australia. On the east is the North Sea fishing and on the west are the immense shoals of herring and mackerel in summer. Some twelve years ago a Scottish woman doctor, with wide experience of children, rather startled a meeting of the British Medical Association held in Belfast by stating that a penny spent on herring was far better value to a growing boy or girl than a penny spent on milk. In the months when herrings are abundant and cheap her contention was, we imagine, perfectly sound. Lastly amongst preserved fish Scotch finnan haddock and kippered herring still reign supreme.

CAMPBELL'S OPERATIVE ORTHOPÆDICS.

THE second edition of "Campbell's Operative Orthopedics" is edited by J. S. Speed and Hugh Smith, both of whom were associated with Willis C. Campbell in the preparation of the first edition in 1939.[1] The first edition was written in a concise style for the benefit of experienced orthopædic surgeons. The second edition, which has been written for the less experienced surgeon, the fellowship man and the house surgeon, is more comprehensive. Because of this different objective much of the text of the original edition has been omitted and a great deal of new material added. The addition of preliminary data and new chapters on pre-operative and post-operative care, peripheral nerve injuries and amputations has necessitated the production of the book in two volumes. New sections have been written on mould arthroplasty by M. N. Smith-Petersen, on ruptured intervertebral disks by F. Murphey, and on difficulty and unusual non-unions by the editors. Many new illustrations have been used, taken largely from journals in which the various procedures have been first presented by the individual surgeons to whom credit is given. The authors of the various chapters are members of the staff of the Campbell Clinic with the exception of Francis Murphey, D. B. Slocum and M. N. Smith-Petersen.

No classification of orthopædic conditions is entirely satisfactory; consequently any arrangement of operative procedures is subject to similar criticism. For example, operations for contracted toes are found in the chapter on static or postural affections, in the chapter on anterior poliomyelitis and also under miscellaneous affections of bone. The operation of capsulotomy of the metatarso-phalangeal joint of the toes which is frequently performed in this country for dorsal subluxation of the toes, so commonly found in static affections of the feet, is described in this book under claw toes of poliomyelitis. However, the provision of an author's index and a subject index with adequate cross indices duplicated in each volume enables the reader to find the operation required.

Operations are clearly described and well illustrated. Many clinical data are included. The authors, however, assume that the reader has had a moderate experience of orthopædic work. This is illustrated by the bald statement that excision of the radial head is the treatment for congenital dislocation of the head of the radius. Undoubtedly in some cases operation is advisable, but in many cases no treatment is indicated.

Whilst this book is not meant to be a text-book of orthopædics and fractures, there are many omissions which, we hope, will be rectified in later editions. In the chapter on difficult non-unions and defect of long bones no mention is made of regaining length by means of distraction in a frame prior and subsequent to bone grafting. The operations of cervical and lumbar sympathectomy are not described. Techniques for stellate ganglion and lumbar paravertebral blocks are not described in detail. In the section on skin plastic procedures there is no description of the use of pedicle grafts, cross leg flaps *et cetera*. Whilst it may be argued that surgery of the sympathetic nervous system is the sole province of the neurosurgeon and that the covering of skin defects is the province of the plastic surgeon, nevertheless, an orthopædic surgeon should be able to deal with a limb threatened with gangrene or cover a skin defect in a compound fracture.

The chapter on affections of muscles, tendons and tendon sheaths is inadequate. This is apparently recognized by the authors who refer the reader to the works of Kanavel, Hart and Bunnell for more detail. Teno-vaginitis of the *extensor carpi ulnaris* tendon, of the peroneal tendons or of the *tibialis posticus* tendon is not even mentioned. Transference of the tendon of the *indicis proprius* to the *extensor pollicis longus* tendon in the treatment of rupture of the *extensor pollicis longus* tendon is not described. The anatomical variations of thumb tendons found in De Quervain's disease are not indicated. Tenosynoviomata, simple or malignant, are not discussed. The author of this chapter does not agree with the modern tendency to conservative treatment of malignant tumours of soft parts, and quite correctly he stresses that when the diagnosis has been established amputation should be performed immediately, despite the possibility of a cure by deep X-ray therapy and excision.

The description of excision of the head of the radius repeats the error of the first edition in describing and illustrating a too extensive excision. One wonders how many cases of inferior radio-ulnar subluxation are seen after the procedure advocated. The insertion of a steel nail after operative reduction of the displaced capitellum in childhood appears to be unnecessary.

This excellent book has been brought up to date, but, for example, the omission of fibular graft or medial tibial graft techniques in the performance of arthrodesis of the ankle illustrates the old axiom that a text-book is out of date as soon as it is published.

We recommend this book to senior house surgeons, fellowship candidates, and general and orthopædic surgeons. It will be found to be an invaluable guide in the operative treatment of orthopædic conditions. As stated in the preface to the first edition, however, the title of this book, "Operative Orthopedics", is not intended to convey the impression that the chief method of treatment of orthopædic affections is operative surgery. The book is a credit to the members of the staff of the Campbell Clinic and a worthy memorial to Willis C. Campbell.

[1] "Campbell's Operative Orthopedics", edited by J. S. Speed, M.D.; Associate Editor, Hugh Smith, M.D.; Second Edition; 1949. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 10" × 6½". Volume I, pp. 912, with 573 illustrations; Volume II, pp. 900, with 568 illustrations, two of which are coloured. Price: £11 5s.

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FAITH.

WE all know that faith, hope and love are named as the three chief virtues and that love is the greatest of them. We admire these virtues when they are displayed by other people, and if we are wise we try to cultivate them ourselves. Practitioners of medicine have reason to contemplate faith, hope and love because of the way in which their work is bound up with the lives—the thoughts and actions—of individual men and women. They are in a position to see, more than any other men with the possible exception of the clergy, the flowering, and not so rare a flowering as some may suppose, of ideal love, the brotherly love of man for his fellow, the love which binds men together and supports them in their relationships one with another. The readiness with which people help one another when trouble arises is a sign of this kind of love, though many who display it would perhaps deny its possession. Incidentally it may be remarked that those who proffer help because of love for a fellow man, should be willing in altered circumstances to accept a gift from another. To refuse is a sign of pride and would cast doubt on the motive in the earlier offering of help. Medical practitioners have seen hope arise in people of all ages—the young, the mature and the aged. They have helped it to start and to grow and have infused it with vigour when it has flagged; they have seen it triumphant, they have seen it deferred, they have seen it shattered. And—incidentally here also—they have been able because of what they have seen and because of what they have sometimes helped to bring about to strengthen their own hopes in life. In the past both love and hope have been discussed in these columns. Faith has been left to the last.

Faith is closely allied to hope. Sometimes hope and faith are almost identical, but generally speaking we may say that faith is the basis of hope. Faith is not easy to define in a few words. We may pass over the cynical definition of H. L. Mencken that it is an illogical belief in the occurrence of the improbable. At the same time there is something in Mencken's use of the word "improbable". We are reminded of this by Sir Thomas Browne, who states in "*Religio Medici*" that to believe only possibilities is not faith but philosophy. It is difficult to think of faith apart from religion; as a matter of fact it is easier to understand in its theological significance. The word faith is applied to the whole body of religious convictions held by the faithful. Apart from this, faith is referred to as an intellectual attribute dealing with the truth of a fact or of an occurrence; and it was Oliver Wendell Holmes who said that faith as an intellectual state was self-reliance. When the will is involved, as in the devotion of self to the principles and practice of religion, faith reaches its highest form. Here it becomes allied to love. We are reminded of this by Dean Inge, who states that faith is an act of self-consecration in which the will, the intellect and the affections all have their place. C. S. Lewis, who is the author of such stimulating books as "The Screwtape Letters" and "The Problem of Pain", writes in "Christian Behaviour" that faith (he is referring to Christian faith) is the art of holding on to things your reason has once accepted, in spite of your changing moods. He states that the moods will change whatever view the reason takes. Lewis as a Christian has moods in which the whole thing looks very unlikely, but when he was an atheist he had moods in which Christianity looked "terribly probable". He holds that it is because rebellion against the real self is inevitable, that faith is such a necessary virtue. "Unless you teach your moods 'where they get off', you can never be either a sound Christian or even a sound atheist, but just a creature dithering to and fro, with its beliefs really dependent on the weather and the state of its digestion." If we are successful in combat against our moods, it is clear that faith becomes a protection and we may remember that Saint Paul described faith as a shield and also as a breastplate. But faith is more than a protection; it gives to life a meaning and a power. Tolstoy said that faith was the force of life and Saint Paul wrote to the Corinthians that we walk by faith and not by sight. As a motive power faith needs to be renewed from time to time. It is something like an electric power supply which needs feeding or reinforcement. Lewis writes that one must train the habit of faith. This is why religious exercises are enjoined on the faithful in religious circles and why they are urged not to forsake the assembling of themselves together. Properly nurtured, faith becomes part of the life of a man and is not readily shed by the well-intentioned. No doubt the Vicar of Bray has his successors; and there are also people to whom the words of Beatrice in *Much Ado about Nothing* would apply. Speaking of Benedick, she said: "He wears his faith but as the fashion of his hat."

What shall we say of faith and the practitioner of medicine? Faith is as necessary to him as to any other person in the community and no further mention need be made of faith in the sphere of religion. A man's religious faith is his own concern, but it may be suggested that it will not be amiss for any man in these days of rapidly changing values to inquire into his own convictions. The doctor has a professional faith on which his life should be based. It is not a religion, but it has tenets analogous to those of a religion, and it is concerned with reverence for human life. At least in his professional life the doctor has consecrated himself to a course, in which, to quote Dean Inge once more, the will, the intellect and the affections have a place. The affections are included, because, though he may not believe in the essential goodness of

man, the practitioner of medicine will be a better doctor if he can treat his fellow man with brotherly love. Faith begets faith. The faith of the doctor in the rightness of his calling and the worth of his work is reflected in the faith of the patient, that he can trust the man who has charge of his physical and mental welfare. Faith triumphs over fear—this for the patient. The doctor needs to remember that "faith without works is dead"—this means Lewis's training in the habit of faith, the refreshment and renewal of faith. In other words there is and always must be a "good fight of faith", not only that faith may accomplish its ends, but that it may be retained as a vital force. In the world of medicine, as in the world of morals and of spiritual welfare, there can be no standing still. Either progress or regression is possible; faith leads to progress and brings life to its fulfilment.

Current Comment.

THE TREATMENT OF STATUS EPILEPTICUS.

THE management of patients with *status epilepticus* is not always understood, but it is a grave emergency and it is important that the best way to deal with it should be known. The occurrence of several probably avoidable deaths from this condition has prompted C. W. M. Whitty and Margaret Taylor,[1] of the Department of Neurology of the Radcliffe Infirmary at Oxford, to review 25 cases of *status epilepticus* and to mention one régime which in their experience has proved a safe and usually effective remedy. The first point that emerges from the study is that in the fatal cases the interval between the onset of *status epilepticus* and the beginning of treatment was usually much longer than in the non-fatal cases, and the duration of *status epilepticus* was longer in the fatal cases than in the others. The implications from this in relation to early and effective treatment are obvious. The second point is that for treatment paraldehyde appears to be the drug of choice, both from the rapidity of its effect in checking convulsions and from its success in abolishing fits in some cases in which barbiturates had failed. The principle of treatment, according to Whitty and Taylor, is to establish as rapidly as possible an effective anticonvulsant level of paraldehyde in the blood and to maintain this until the fits stop. They recommend the following régime for an average adult. As soon as possible eight to ten millilitres of paraldehyde (which need not be sterilized) are injected into the gluteal muscle, and the site of injection is massaged; this treatment, it is stated, usually stops the fits within half an hour. If the fits continue, five millilitres of paraldehyde are given intramuscularly every half hour until the fits cease; the persistence of focal twitching without any tendency to spread is not an indication for further sedative, and attempts to eliminate these entirely may lead to a dangerous level of narcosis. If the patient's general condition indicates it, intravenous drip therapy with glucose and saline solution or plasma is instituted and maintained at the rate of one bottle in three hours; this will also provide a convenient method of continuing the administration of paraldehyde, either by intermittent injections into the drip tubing or in solution in the drip fluid in any required concentration, since it is soluble in a dilution of one in eight in physiological saline solution. Intravenous drip therapy is not always required, but it is useful as a means of adjusting accurately the blood paraldehyde level so that fits are controlled but coma is not too deep; it may be necessary to combat dehydration or the accumulation of metabolites; and it is obviously indicated as the medium for paraldehyde administration when the patient's circulatory state suggests that absorption will not be effective from intramuscular injection. For children the dosage is smaller, but it is still higher than is generally realized, and even for infants aged only a few months two or three millilitres of paraldehyde must be given intramuscularly to be effective.

The relative safety and rapid action of paraldehyde make it the drug of election, but if it is not available, an attempt must be made immediately to stop the fits by other means. If phenobarbitone is used, it should be given in doses of six to twelve grammes, preferably in the "soluble" form by the intravenous or intramuscular route. Thiopentone in anæsthetic doses may be used, or chloroform anæsthesia if no other remedy is at hand. However, the effect obtained by these methods is often short-lived. The respite given by the immediate measures is probably best used in establishing paraldehyde sedation.

Once *status epilepticus* has been controlled, regular anticonvulsant therapy, for example, in the form of the barbiturates or hydantoin, must be instituted and the patient made to understand that the regular use of these medicaments for an indefinite period is essential to avoid recurrence of the convulsions. The omission of the patient's usual anticonvulsants in either symptomatic or idiopathic epilepsy is a common precipitating factor of *status epilepticus*, and this cannot be stressed too much, especially to patients apt to be careless about their regular medication; although early and adequate treatment is quite often effective and although occasionally *status epilepticus* seems to subside practically without treatment, every case has to be regarded as potentially fatal.

[1] *The Lancet*, October 1, 1949.

BACTERIÆMIA AFTER TOOTH EXTRACTION.

THE occurrence of transient bacteriæmia after tooth extraction and even after much milder disturbance of the teeth was mentioned in these columns on May 1, 1948. The serious significance of this fact was recognized in relation to bacterial endocarditis, and we referred to the encouraging results obtained with penicillin in the control of the bacteriæmia. Interesting additional points about the bacteriæmia have come to light in a report by M. G. McEntegart and J. S. Porterfield[1] of the investigation of 200 patients who underwent tooth extraction. Blood samples were collected before and after the extraction. With one exception—in which a diphtheroid organism was recovered—all pre-operative specimens were sterile. Organisms were grown from the post-operative specimens in 113 cases (56·5%). Streptococci predominated, but diphtheroids were grown in five cases and obligatory anaerobic Gram-negative cocci in six cases. The latter were all found in the second hundred of the 200 cases, and the organism may have been present in the first hundred and its significance not appreciated; it was identified as *Veillonella gazogenes*, which has been recovered from normal sputum and, much more important, was found by L. Loewe, P. Rosenblatt and E. Alture-Werber[2] to be the cause of a case of subacute bacterial endocarditis which proved refractory to treatment and was arrested only by a combination of penicillin, sodium para-aminohippurate and heparin. This organism is not recovered by the usual technique of blood culture in bottles of nutrient broth, and it is suggested that attention to anaerobic methods may reveal it as the cause of other infections; perhaps it will explain certain cases of subacute bacterial endocarditis in which the clinical picture is characteristic, but repeated attempts to recover an organism from the blood are unsuccessful. Another interesting aspect of the investigation was the correlation of the incidence of streptococcal bacteriæmia with the state of the gums, the number of teeth extracted and the duration of the operation. First perusal of the results obtained suggests that the amount of gingival disease directly influenced the incidence of bacteriæmia, but this appears to be fallacious; more careful analysis indicates that the incidence of

[1] *The Lancet*, October 1, 1949.
[2] *American Heart Journal*, September, 1946.

bacteriæmia rises with the number of teeth extracted (that is, with the amount of trauma inflicted) and is not likely to be greater in a patient with severe gingival disease than in one with healthy gums who is subjected to the same number of extractions. A limited series of observations supports the view that the duration of the operation also is more important than the state of the patient's gums in determining the incidence of bacteriæmia. Experiments in which a non-pathogenic organism was applied to the gums before teeth were extracted indicate that any organism applied to the gum margin may enter the blood-stream as a result of tooth extraction, irrespective of the severity of infection prevailing in the mouth. These findings are in line with previous work and reinforce the emphasis which has been placed on the need for caution in approaching dental extraction in the presence of cardiac defects of congenital or rheumatic origin; the use of prophylactic measures should at least be considered. Two particular points brought forward are, first, the fallacy of trusting in the apparently healthy state of the gums and overlooking the importance of the extent of the trauma and the duration of the operation, and second, the need to bear in mind *Veillonella gazogenes*, which may elude discovery and can cause infection refractory to ordinary therapeutic measures.

PSYCHOLOGICAL ASPECTS OF PÆDIATRICS.

FOR some time Harry Bakwin and Ruth Morris Bakwin, with occasional contributions by other writers, have been presenting a series of articles on various psychological aspects of pædiatrics in *The Journal of Pediatrics*. Brief reference to these articles may be of general interest. In a recent paper Harry Bakwin[1] discusses the gifted child and reviews a survey of 1500 gifted Californian children from 1921 to 1945. He states that this group proved to be superior to the general population in health and physique, versatile rather than one-sided in achievement, and superior in personality and emotional stability; they enjoyed a higher marriage rate and better marital adjustment. An important finding is that at school gifted children who have been promoted rapidly are better in health and general adjustment than if kept back. Contrary to a widespread impression the survey indicated that the intelligence of their children tends to regress to the mean. Coming to the other extreme of childhood intelligence, Ruth and Harry Bakwin[2] discuss the child with mental deficiency. They point out that many children with lesser degrees of mental deficiency become self-supporting, doing unskilled work, but becoming socially well adjusted and supporting a family, in which the children's intelligence, like that of the children of the gifted group, tends to approach the mean. It is urged that the parents of mentally defective children should be told clearly that the child is backward, but that he will learn slowly; the intelligence quotient gives an idea of how fast and how much the child will learn. It is important also to appreciate that the child is not unhappy because of his handicap. Training and institutional care are discussed in this paper. The use of glutamic acid in mental deficiency is the subject of separate consideration by Harry Bakwin.[3] He states that it does cause some rise in intelligence quotient while it is being given, and seems worth while for the child who is a little backward, as it may help him to cope better with his environment and so keep up with his fellows. It gives hope to the parents and so improves parent-child relationships, thereby lessening emotional disturbances, which so often exaggerate the plight of the slow child.

The child with asthma may present a particular problem of his own. He is described[4] as often alert, over-anxious and irritable, with over-anxious and over-protective parents. Some children accept the pampering of the mother and become dependent, but others resent it and become antagonistic and disobedient. These factors should be taken into account in the management of the asthmatic child, but they do not preclude other forms of therapy.

Thumb and finger sucking Harry Bakwin[1] regards as a harmless habit. He quotes authorities to support his view that it is a much less harmful influence on dental alignment than is heredity. He believes that throughout life the mouth is used for pleasure, and thumb sucking in the infant is no more undesirable than gum-chewing in childhood and smoking and munching in adult life. He urges that the harmless nature of the habit should be explained and methods of restraint abandoned.

Breast feeding is discussed by Harry Bakwin and Samuel Stone.[2] The advantages of breast feeding from the point of view of quality of protein and fat, nitrogen retention, carbohydrate, mineral and vitamin absorption, and resistance to infection are considered in detail, and the relative freedom of the breast-fed baby from diarrhœa and eczema is mentioned. It is pointed out that from the psychological aspect breast feeding provides emotional satisfaction to both mother and baby, and while no claim is made that all breast-fed babies will grow into happy adults or *vice versa*, it is felt that breast feeding is an important step in establishing proper interrelationships between mother and baby and in providing suitable outlets for the baby's budding emotional needs. Bakwin and Stone do not minimize the enormous value of artificial feeding when an honest attempt at breast feeding has failed. Another problem of feeding, the occurrence of a poor appetite in the young child, is dealt with by Harry Bakwin.[3] He discusses the factors contributing to feeding difficulties in the toddler: the diminished growth rate at this age, the emerging self-dependence of the child and his resistance to compulsion, the insistence of parents on certain foods considered essential though unpalatable, the continuation of "puréed" foods after the child is ready and willing to chew, the failure to allow the child to feed himself when he begins to show an interest in this new achievement, and sometimes the presence of physical ill-health or serious emotional disturbance. Food should be attractive, suitable for the child's age and to his taste, and it must be appreciated that most children like seasoned food. It is better to forget Calories, vitamins and minerals, and the child should feed himself. Discussion of eating in the child's presence, bribing, compulsion and excessive antics at meal times should be avoided. However, we all eat better in good company, and a certain amount of entertainment for the child at meal times is probably desirable. If a deep-seated emotional problem is present, investigation of the whole situation is necessary. Tonics and vitamins should be avoided, for they are unnecessary and divert the parent's attention from the real mechanisms underlying the feeding difficulty.

There are a number of other papers in the series on which comment might be made, but perhaps it will suffice to mention them. Organic cerebral damage underlying behaviour disorders in children is described by Harry Bakwin;[4] encephalitis, burn encephalopathies, head injury, brain tumours, cerebral anoxia, pertussis and allergy are considered. Ruth Bakwin[5] details the indications for and types of tests used in the mental testing of children. She also looks into[6] the problem of the blind child from the point of view of mental functioning, personality, education, occupation and care. Pure maternal over-protection, analysed by Harry Bakwin,[7] the benefit of the nursery school to the child, described by Barbara Biber,[8] the summer camp, considered by Cornelia Goldsmith,[9] and psychosomatic illness in the child, discussed by Walter C. Price,[10] are further interesting subjects in a series, which should be of use to all practitioners who are concerned with the psychological problems of children.

[1] *The Journal of Pediatrics*, August, 1949.
[2] *Ibidem*, May, 1948.
[3] *Ibidem*, December, 1947.
[4] *Ibidem*, March, 1948.
[1] *Ibidem*, January, 1948.
[2] *Ibidem*, November, 1948.
[3] *Ibidem*, November, 1947.
[4] *Ibidem*, March, 1949.
[5] *Ibidem*, September, 1948.
[6] *Ibidem*, July, 1949.
[7] *Ibidem*, December, 1948.
[8] *Ibidem*, January, 1948.
[9] *Ibidem*, April, 1949.
[10] *Ibidem*, June, 1949.

Abstracts from Medical Literature.

THERAPEUTICS.

Lead Poisoning.

W. C. WILENTZ (*The Journal of the American Medical Association*, March 20, 1949) describes the treatment of lead poisoning. He recommends the administration in the acute stage of half an ounce of magnesium sulphate every morning, a diet of high calcium content, 10 millilitres of 20% calcium gluconate solution by the intravenous route every four hours, and for colic antispasmodics such as belladonna, phenobarbitol and trasentin hydrochloride. The author discusses "deleading" by drugs such as potassium iodide, ammonium chloride, BAL and parathyreoid hormone. He shows that there is disagreement as to whether these drugs actually do cause liberation and excretion of lead, but fairly general agreement that their action is dangerous. There is not universal acceptance of the view that calcium causes retention of lead in the bones, but this view is commonly held, and it is said that the surplus lead is gradually excreted under a calcium régime. Intravenous calcium therapy is continued for three or four days, and then calcium wafers are given by the mouth. Vitamins, milk and special diets are acceptable, but their value is questionable.

Primary Atypical Pneumonia.

G. MEIKLEJOHN AND R. I. SHRAGG (*The Journal of the American Medical Association*, May 28, 1949) discuss the use of aureomycin in primary atypical pneumonia. Patients selected for treatment were those whose symptoms and signs suggested primary atypical pneumonia, whose X-ray films showed evidence of pneumonia, whose temperature was above 102° F. for twenty-four hours before treatment, whose white cell count was less than 12,750 per cubic millimetre, and whose condition was not showing improvement. Fifteen patients were treated with penicillin and fifteen with aureomycin. Fifty milligrammes of aureomycin in five millilitres of a leucine diluent by intravenous injection and an oral dose of one gramme were given initially. Thereafter patients received one gramme by mouth every six hours until the temperature had been normal for forty-eight hours. The patients treated were as far as possible comparable, so far as signs and symptoms and results of laboratory investigations were concerned. Two-thirds of the penicillin-treated group recovered slowly, fever lasting in two cases for twenty-two and twenty-three days respectively. One-third of the patients recovered in forty-eight hours. This is said to be the usual course of events in primary atypical pneumonia. The aureomycin-treated group on the other hand showed definite symptomatic improvement within three days or less, and became afebrile within five days. Aureomycin therapy was continued for two to five days after the temperature had become normal. The clinical and radiological signs decreased more slowly than the symptoms, though the physicians thought the recovery was more rapid than expected. Three patients relapsed from three to six days after aureomycin therapy was suspended. Fever and radiological signs increased and white cell counts of 15,000 to 29,000 per cubic millimetre were observed in two cases. Penicillin treatment was given to these two patients, but they did not improve until aureomycin replaced penicillin. All three became afebrile within twenty-four hours of the reinstitution of aureomycin therapy. Cold agglutinins in significant titre were demonstrated in 12 of 20 patients in both groups. Three patients whose condition became worse on penicillin treatment were changed over to aureomycin, with pronounced and rapid improvement.

Hypertension.

W. M. CRAIG (*The Journal of the American Medical Association*, April 30, 1949) publishes an evaluation of the treatment of hypertension. He quotes Wagener and Keith's classification of essential hypertension into four groups—slight to moderate with 30% to 42% deaths in four years, moderately severe and severe with death rates of 78% and 98% in four years. The author advises preventive measures among patients with an hereditary taint who have any sign of hypertension. He suggests guiding their activities, and their intake of food, alcohol and tobacco. Medical treatment consists of rice or low sodium diet, thiocyanates, "Rutin", barbiturates, and *Veratrum Viride*. Nitrites, acetyl choline, histamine and tetraethyl ammonium chloride or bromide have a transient effect. Sedatives are the most valuable drugs. If medical treatment is not satisfactory surgical treatment is advocated. A great difficulty is encountered in the selection of suitable subjects for operation. Patients with fixed hypertension associated with diffuse arterial sclerosis are not benefited by operation, and severe hypertension in the later stages does not respond with permanent benefit. Irreversible changes in the vascular, muscular, nervous, cardiac or renal systems are a contra-indication to operation. The effects of rest in bed, of sedation and of tetra-ethyl-ammonium chloride or bromide are to some extent helpful in deciding upon operation. This involves operation on the sympathetic nervous system to relieve vasomotor spasm of the vessels of the splanchnic region, abdomen and lower limbs. There appears to be no doubt that the surgeons prefer to operate on patients whose hypertension is in the early stages and for whom the prognosis from the medical aspect would be in many cases fairly good.

Sympathicolytic Drugs in Hypertension.

M. P. ROGERS (*The Journal of the American Medical Association*, May 21, 1949) discusses drugs for reducing blood pressure in the hands of the general surgeon. Tetra-ethyl-ammonium bromide and chloride and "Dibenamine" (dibenzyl β chloroethelamine hydrochloride) will block effector sympathetic pathways, but must be used intramuscularly or intravenously. "Priscol" (2-benzyl-4,5 imidazoline hydrochloride) can be used orally. Doses of 20 to 50 milligrammes every two to four hours given intramuscularly or intravenously are safe, except in coronary and cardiac disease. However, oral administration was effective in doses of 25 milligrammes five times daily, in pain due to peripheral vascular disease. Arteriosclerosis, diabetic arteritis, Buerger's disease and thrombophlebitis with pain in the legs responded to "Priscol" given intravenously or orally. Oral treatment with 25 milligrammes four or five times daily was continued for weeks or months, relieved pain, and was often considered to assist in recovery. Two patients with hypertension were treated with "Priscol" or "Priscoline", 50 to 75 milligrammes every two and a half hours, with considerable lowering of blood pressure and without ill effects. One patient took the drug for eleven month. In *tabes dorsalis* and syphilitic gastric crises also pain was relieved by "Priscol".

Essential Hypertension.

R. W. WILKINS, E. D. FREIS AND J. R. STANTON (*The Journal of the American Medical Association*, May 21, 1949) discuss drugs used in the treatment of essential hypertension. *Veratrum Viride*, especially a new standardized preparation "Vertavis" reduces blood pressure in emergencies, but has a tendency to cause nausea, vomiting and even collapse. Laboratory tests showed that veratrum acted as a general vasodilator, causing increased cardiac output and oliguria. The investigations did not clearly reveal whether the drug was sympathicolytic or parasympathicomimetic, but its action was through the nervous system. Dihydroergocornine had a clear-cut sympathicolytic effect, and reduced blood pressure without causing tachycardia. The doses used were 0·7 millilitre of "Veratrone" given intramuscularly, and 0·4 millilitre of dihydroergocornine given intravenously; but the authors state that the drugs are used orally and cause prolonged fall of blood pressure in encephalopathy or myocardial failure secondary to severe hypertensive crises.

AN EDITORIAL NOTE (*ibidem*) states that further studies have shown that patients became resistant to dihydroergocornine after long-continued oral administration.

Management of Neurosyphilis.

C. EARLE JOHNSON, JUNIOR (*The Journal of Nervous and Mental Disease*, May, 1949) has decided that until treatment for neurosyphilis by penicillin has been standardized it is advisable to combine it with the older techniques. Treatment is individualized within the framework of four phases: penicillin therapy, malaria treatment, active chemotherapy and the follow-up stage with periodic serological examinations. The author commences treatment with penicillin on orthodox lines (three to six million units in ten days) followed by malarial therapy (twelve paroxysms of quartan malaria terminated by "Atebrin" sulphate). The treatment by chemotherapy varies with the type of syphilis. For paretics tryparsamide is given in four courses during the following two years. Patients with *tabes dorsalis* have three courses. In both a series of injections of oil-soluble bismuth alternates with those of tryparsamide. The technique for combined cardio-vascular syphilis and neurosyphilis includes preliminary courses of "Mapharsen" and sodium bismuth tartrate. The author confirms the danger of amblyopia in tryparsamide medication, but considers that this risk does not preclude its use. In every case the visual fields should be

tested before treatment. By this means it will be shown that some examples of amblyopia are present before treatment is begun, and do not result from it. For emesis occurring with tryparsamide he uses the intravenous injection of calcium gluconate (one gramme to ten millilitres of distilled water) just before the drug is administered. With regard to the rationale of cessation of treatment, the first and most rigid requirement is a minimum of two years' continuous post-malarial chemotherapy. In cases of persistent positive serological findings after treatment, a rest of three to twelve months is given, followed by a further six to twelve months of additional chemotherapy. The physical, mental and serological findings are important in assessing the need for further treatment.

NEUROLOGY AND PSYCHIATRY.

Electroencephalogram in Scleroderma.

R. M. TAYLOR AND B. L. PACELLA (*The Journal of Nervous and Mental Disease*, January, 1949) discuss the previously noted relationship between lesions of the central nervous system and scleroderma. They record the electroencephalogical findings in 56 cases; 84% of patients had abnormal patterns, mostly of slow (32%) and convulsive (21%) types. The degree of abnormality was unrelated to the severity of the disease. The authors suggest that decrease in the amount of ionized calcium in the fluid environment of nervous tissue may be a factor in production of abnormality.

Hypnosynthesis.

JACOB H. CONN (*The Journal of Nervous and Mental Disease*, January, 1949) states that modern psychiatry began only when the patient began to participate in his own treatment. He attempts to bring hypnosis into this field by a technique of less depth of hypnosis, although previously it was considered that hypnosis must be deep to be effective. A trance state is induced. The patient is carefully told how he must act, but is told not to fall to sleep; he will be relaxed and able to discuss his case freely. Patients have been under treatment up to two years. Success has been achieved in difficult cases. The author claims that the method can be used therapeutically for active patient-participation as a creative, unifying, interpersonal experience, and therefore can be classified as a form of dynamic psychotherapy.

Convulsive Therapy.

PETER A. MARTIN (*The Journal of Nervous and Mental Disease*, February, 1949) discusses the results in 511 cases of convulsive therapy since 1939. Six deaths occurred during but not through the treatment. The author believes that there is evidence of petechial hæmorrhage present in some cases. He concludes that shock is of definite value in a State hospital and is least useful for patients with schizophrenia. He warns that shock therapy should be used neither indiscriminately nor by anyone who has access to a shock machine. Considerable actual bedside shock experience is necessary before the therapist learns when and how to use shock therapy. It should be one of several skilled tools which the therapist has at his command and not the entire therapeutic armamentarium. With experience, the therapist can use shock judiciously in cases in which it has been previously contraindicated. Also it should be judiciously withheld in selected cases of psychotic withdrawal when continued therapy causes further regression or a shock psychosis. This can be learned only by actual clinical trial. In short, with shock patients, as with all others, the therapist must constantly consider the individual patient as he decides how to put this valuable tool to its best advantage in each case.

Psychological Significance of Restraint.

M. H. ERIKSON (*The American Journal of Psychiatry*, February, 1949) draws attention to the necessity of considering physical restraint of mentally disturbed patients as often fulfilling a desire or giving an opportunity to express in some physical way the unknown unrealized personality conflict of the psychotic patient. He points out that comfort and security can be afforded to insecure children and frightened patients by tight and restricting arms, or by crowding together. He gives a number of examples from twenty years' experience in which patients have asked to be placed in restraint, particularly those that are subject to periodic outbursts of maniac-like behaviour. In these cases it has been found that a relatively short period of this form of treatment in the initial stages of an outburst will ultimately result in the acute episode's passing off rapidly. It is not a plea for more restraint, but rather that it should be realized that restraint can be of therapeutic benefit to the patient.

Sequelæ of Lobotomy.

AUGUSTUS S. ROSE (*The Journal of Nervous and Mental Disease*, March, 1949) discusses routine neurological examinations made on 360 patients subjected to lobotomy. Although the operation is sometimes performed for the relief of painful metastases, there is frequently a painful response to skin stimulation. The reaction to pin-prick on the sole is excessive. The author notes a disturbance in the control of bladder function. Although the patient knows he is urinating and can control the function, he persists in it without shame. It is inferred that lobotomy interrupts the mechanism of acquired automatic control.

Birth Injuries and Mental Deficiency.

L. S. PENROSE (*The Journal of Mental Science*, April, 1949) states that there is an ætiological distinction between symmetrical and asymmetrical congenital plegias, and that it has become a general rule to confine the diagnosis of trauma to asymmetrical lesions. The familial incidence is low, not amounting to more than one in twenty among siblings of the patient; the fact that the rate is increased in consanguinity suggests strongly that some cases are due to rare recessive genes. It is difficult to believe that a traumatic hæmorrhagic lesion can often be symmetrical; however, necrosis of cells due to asphyxia, prenatal disturbances or disease may produce such an effect. Discovery of the cause of the defect in any one case necessitates a complete family and personal history, it being remembered that prematurity, long labours, primiparous births and asphyxia (for example, following Cæsarean deliveries) are predisposing causes of hæmorrhage, and that pallor, tension of the fontanelle, convulsions and difficulty in feeding are signs suggesting traumatic damage.

Crushing Injuries to the Skull.

W. RITCHIE RUSSELL AND F. SCHILLER (*Journal of Neurology, Neurosurgery and Psychiatry*, February, 1949) discuss crushing injuries to the skull. This work is based on the analysis of clinical head injuries and on experiments in which forces were applied to a skull in different directions by means of a clamp. The pattern of the fractures, as they affect the base of the skull, is described. The authors suggest that it is often difficult to obtain X-ray evidence of fractures of the base of the skull.

Combined Therapy.

SELIG M. KORSON (*The Journal of Nervous and Mental Disease*, January, 1949) discusses the difficulty in treating severe obsessive compulsive states. He describes a case history of an intelligent white male whose whole life revolved around sexual phantasies and actions to such an extent that he was treated in an institution for six years. At the end of this time narcosynthesis with "Sodium Amytal" (seven and a half grains) given intravenously was employed in a two-hour session for fifty weeks. The symptoms lessened, but did not disappear. Electroshock, thirteen daily sessions, was then employed in the hope that the amnesia would be beneficial but would not destroy the strong transference achieved in a year of narcoanalysis. An acute episode followed the electroshock; it was succeeded by insight and freedom from symptoms. The case history is a reminder of the need for persistence in some cases.

Intensive Electroplexy.

MAX VALENTINE (*The Journal of Nervous and Mental Disease*, February, 1949) has drawn attention to the need for knowledge as to the optimum amount of electroplexy in various cases. On a basis of clinical experience he considers that intensive electroconvulsive therapy of the daily or even twice daily variety is sometimes extremely useful in unpromising cases. The cumulative phase of electroplexy is speeded up. It is particularly helpful in acute psychosis with refusal of food, acute mania, the paranoid type of involutional psychosis and paranoid psychosis.

Inducing Anxiety.

EDMUND F. WALKER (*The Journal of Nervous and Mental Disease*, March, 1949) believes that psychotherapy is essentially a reeducation in the problems of environment and self. He considers that it is useful to be able to create anxieties in order to demonstrate their rationale and to assist in the unearthing of repressed material. Three cases are quoted in which the therapist deliberately created conditions of anxiety which had beneficial results in creating a more mature adjustment to environment.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on June 1, 1949, in the Medical Society Hall, East Melbourne, DR. DOUGLAS THOMAS, the President, in the chair.

The Use of Anticoagulants.

DR. W. McI. ROSE read a paper entitled "Experience in the Use of Anticoagulants" (see page 665).

DR. PAUL FANTL read a paper entitled "The Use of Anticoagulants" (see page 667).

DR. GEOFFREY NEWMAN MORRIS asked for information on the safety of the use of anticoagulants in nursing homes or small hospitals for several days without control of the prothrombin content of the blood.

Dr. Rose replied that he did not like the practice, even though no harm might come of it six times out of seven. It was probable that ethylidene dicoumarin could be used in those circumstances in the dosage he had outlined for four or five days, but that was not long enough to use it; the treatment should be continued until the patient was ambulatory. It was not really safe to use anticoagulants without knowledge of the prothrombin level, but it might be better than doing nothing. He had heard that, in Sydney, heparin was at times used for two days followed by dicoumarin for three days, and then treatment was stopped; that appeared to him to be a "hit or miss" procedure, but it was better than nothing.

DR. H. B. KAY said that at the Alfred Hospital he had found that, with the administration of ethylidene dicoumarin, the level of prothrombin dropped quickly, but rose again within two days; it fell again and stayed down on continuation of the treatment. He added that no drop in the level could be obtained sometimes, and that it had been reported from Sydney that dicoumarin would not consistently lead to a fall in prothrombin level.

Dr. Rose, in reply, agreed that in cardiac infarction an unexpected drop in prothrombin level was apt to occur when ethylidene dicoumarin was used. It might be ascribed to peripheral cardiac failure with impoverished metabolism. He had encountered the phenomenon, but not often. In one case, despite the use of 300 milligrammes, there was a rise to 100% in the prothrombin level, and the patient suffered a secondary coronary thrombosis.

DR. JOHN HAYDEN said that in his limited experience, in some cases of coronary occlusion the prothrombin level had fallen much more rapidly than was desired. In one example the prothrombin level was at 100% when dicoumarin was given, 500 milligrammes being followed on the same day by 100 milligrammes; by the next day the prothrombin level was only 12%. It seemed that in cardiac infarction the level could readily be reduced; as a result they were using smaller dosage in those cases than was usually recommended. With reference to the diagnosis of pulmonary embolism, Dr. Hayden said that a sudden post-operative rise of temperature—an isolated peak or spike—frequently occurred three or four days before a fatal embolism; that was a valuable diagnostic sign.

Dr. Rose, in reply, said that he was not sure of the dosage in cardiac infarction; the prothrombin level might drop rapidly—perhaps by as much as 50%. If on that account a smaller dose was used, the protection desired might not be obtained. He went on to say that he had seen no harm follow a fall to 10% on the third day; if the aim was to prevent thrombotic complications he felt that it was best to give the standard dose and then to wait and see what happened.

DR. MORRIS DAVIS said that, in coronary cases, when ethylidene dicoumarin was used the prothrombin level was often low after the coronary episode, and he had learned to be conservative if he could not have the prothrombin level determined before starting to use the drug. He asked Dr. Rose whether he concurred in that attitude. He also asked when treatment with anticoagulants should be stopped in venous thrombosis of the legs.

Dr. Rose, in reply, said that he found it difficult to confirm Dr. Davis's attitude, because as the normal range was so wide, varying between 60% and 140%, what appeared to be normal might be actually a low level in an individual case and *vice versa*. In reply to the second question, Dr. Rose said that he considered it was advisable to continue treatment until the patient was up and walking with the leg in an elastic bandage; that was usually for three weeks.

DR T. E. LOWE said that he was puzzled by the whole story. Though he had had no direct clinical experience of the use of the anticoagulants in man, he had used heparin and dicoumarin in dogs, but the dosage required to achieve results was enormous; feeding dicoumarin to dogs for days before the operation was a waste of time—it would not prevent clotting. He believed that it must be questionable whether the anticoagulants were acting directly on the clotting mechanism at all; they should look for some other action.

Dr. Lowe then said that another puzzle was the clinical application of the therapy. Apparently it was of undoubted value in pulmonary infarction, but in the other fields its clinical value remained to be proved. It was obvious from published series that personal factors were influencing the reports. One thing had emerged from the work done which was important—it was clearly proved that dicoumarin was a dangerous drug. In reports from abroad it had been used without adequate controls, and many deaths from hæmorrhage had resulted; hæmorrhage from dicoumarin therapy was hard to check. In his experience, no two laboratories could duplicate results.

DR. DOUGLAS THOMAS said that in the old days they had formed the concept of a "trigger area" acting as a focus from which the metastatic phenomena spread or emanated. If that concept was no longer valid, he thought that they must consider the rate of flow in any area; such factors as age might initiate a local event. He, too, knew of wide discrepancies in laboratory results. He quoted an instance in which inadvertently the prothrombin level fell to below 5%, and despite cessation of anticoagulant therapy remained at that low level for fourteen days; yet no hæmorrhages occurred. He himself had an increased feeling of security when the anticoagulant therapy was controlled by estimations of the prothrombin level.

DR. RODERICK ANDREW referred to two initially low prothrombin readings—20% and 30% respectively—which had been cross-checked in two laboratories. He also mentioned a practical point on prescribing anticoagulants: ethylidene dicoumarin was ordered, but dicoumarin was supplied; disastrous results might follow if the error was not noted in time.

DR. E. E. DUNLOP asked whether Dr. Rose had observed that the sensitivity to anticoagulants was influenced by the duration and magnitude of the operative procedure. He had noticed that clotting complications were much commoner after lengthy operations such as œsophagectomy, and yet those patients were unduly sensitive to anticoagulants; he had used half-dosage with falls of prothrombin level to 10% and 5% and with hæmorrhages in some cases.

Dr. Fantl expressed agreement, and added that the mechanism depended also on the anæsthesia; prolonged administration of ether led to a drop in the prothrombin level.

DR. HOWARD EDDEY said that, though his experience was limited, he had on a number of occasions used ethylidene dicoumarin in medical cases and usually in private houses. The laboratories had been cooperative and in most cases the prothrombin levels were in the 30% group. A patient with coronary occlusion and very severe pain was rapidly relieved by ethylidene dicoumarin. Dr. Eddey believed that such results were a justification for the use of the drug even without laboratory control.

Dr. Rose, in reply, said that he was afraid to give the drug without control; there had been many tragedies in the United States of America with uncontrolled use of the anticoagulants. The difficulty was to have the advantage of laboratory control for patients treated in their own homes. The tendency was to give the treatment and to hope that all would be well; but Dr. Rose deplored that approach to the problem, though he admitted that there were circumstances in which the patient was not receiving the best care unless some such risk was taken. In the three public hospitals with which he was associated, Dr. Rose had found great constancy in the laboratory results; variations might be due to lack of experience. He was not in a position to discuss the points raised by Dr. Lowe, as he had had no experience with dogs and was unaware of their reactions to the drugs. As far back as 1928, a series of cases was reported of thrombotic complications of cardiac infarction in which the prothrombin level was as high as 45%.

DR. IAN WOOD said that the papers and the discussion were valuable contributions to the knowledge of the subject. They should all aim at efficient observation of the prothrombin levels. He asked what was the prognosis when the hæmorrhagic state was established.

Dr. Rose, in reply, expressed the opinion that the gravity of the prognosis was decreased. An earlier dose of vitamin *K* was inadequate; the prothrombin level in hæmorrhagic cases must be raised above a certain critical point; the ordinary preparation containing five milligrammes of vitamin *K* in oil was effective in infancy, but, for the adult, 60 milligrammes might have to be given as often as every two hours. He said that Nicholas, Proprietary, Limited, had made available a 10-millilitre ampoule containing 60 milligrammes of water-soluble vitamin *K*, but, of course, transfusion of blood helped to control hæmorrhage.

DR. JOHN HAYWARD said that in thoracoplasty and pulmonary resection operations he had had no trouble with post-operative thrombosis. He wondered whether it was possible that operative interference with the lung had some effect on prothrombin production. In œsophagectomy and removal of carcinomata of the lung they had great trouble from thrombosis; yet in operations of comparable magnitude, as he had mentioned, that was not the case. He asked Dr. Rose what to do if he had a patient with carcinoma receiving ethylidene dicoumarin, whose prothrombin level was 30%, and yet thrombosis actually occurred.

Dr. Rose said that heparin should be given. There would occur some cases of thrombosis in the 30% to 50% group, but in that range it was unlikely to prove fatal.

DR. G. HARKNESS also expressed a warning against using dicoumarin without laboratory control. He said that he was surprised at the varying response in different cases to similar doses. One patient's prothrombin level dropped to 25% after a dose of 500 milligrammes, but the next patient's prothrombin level might drop to 12% and stay there for three days without additional doses. He deprecated the use of the drug without the necessary control.

DR. LORNA LLOYD-GREEN asked Dr. Rose whether the prophylactic administration of anticoagulants was dangerous in eclampsia or in the presence of hepatic or renal damage during pregnancy.

Dr. Rose said that, at the Women's Hospital, the therapy was used for all toxæmic patients and for those with carcinoma and those requiring Cæsarean section; but there was no evidence that the toxæmic patients became more sensitive to the drugs. An eclamptic patient needing the Cæsarean operation had a grave risk of femoral thrombosis and should be given ethylidene dicoumarin. Dr. Rose said that the drug passed to the breast milk and thus to the child and might lead to hæmorrhage. The children, especially if premature, should be given "Campolon" and vitamin *K*, but that was not necessary if the baby was at full term and was vigorous. Dicoumarin administered before delivery had not, in his experience, affected the child.

A MEETING of the New South Wales Branch of the British Medical Association was held on September 22, 1949, at Sydney Hospital. The meeting took the form of a series of clinical demonstrations by members of the honorary medical and surgical staffs of the hospital and by the almoners' department.

Thyreotoxicosis.

Thyreotoxicosis Treated with Propyl Thiouracil.

DR. E. H. STOKES first showed a young, unmarried woman, aged eighteen years, who was suffering from thyreotoxicosis. She had first noticed a swelling in her neck two years previously. She had suffered from occasional choking sensations, frequent attacks of weeping and numerous bouts of palpitation. She had lived in Sydney all her life and had suffered from rheumatic fever at the age of four years. Her mother, aged forty-nine years, was stated to be suffering from hypothyreoidism.

On examination of the patient, it was noted that she was somewhat obese. The eyes were prominent and the palpebral fissures widened. The thyreoid gland was considerably enlarged and was firm in consistency. A bruit was audible over both lobes of the gland. The first heart sound in the mitral area was split. The pulse rate was 120 per minute and the blood pressure readings were as follows: systolic, 170 millimetres of mercury, and diastolic, 100 millimetres. There was slight tremor of the hands. The basal metabolic rate on August 2, 1949, was +40% and the serum cholesterol content was 64 milligrammes per 100 millilitres.

Treatment with propyl thiouracil had been instituted early in August, and she was receiving 0·6 gramme daily at the time of the meeting. Her general condition had improved, the pulse rate having fallen to 80 per minute; but the thyreoid gland was still enlarged. Dr. Stokes said that it was proposed to watch the patient's progress for some time before deciding that thyreoidectomy should be performed.

Thyreotoxicosis Treated with Methyl Thiouracil.

Dr. Stokes next showed two patients suffering from thyreotoxicosis, who had been treated with methyl thiouracil.

The first patient was a married woman, aged twenty-nine years, in whose case the symptoms of thyreotoxicosis had commenced two and a half years previously. After parturition, she had become tremulous and excitable. In January, 1949, the patient's mother noticed that her eyes had become prominent. Since that time she had suffered from palpitation, dyspnœa and excessive perspiration.

On examination of the patient on June 23, 1949, it was seen that she was thin and nervous. The eyes were prominent and the thyreoid gland was moderately enlarged and soft. The palms of the hands were moist. The pulse rate was 120 per minute and the blood pressure readings were as follows: systolic, 190 millimetres of mercury, and diastolic, 120 millimetres. Glycosuria was present. The basal metabolic rate was +44% and the serum cholesterol content was 110 milligrammes per 100 millilitres.

The patient was treated by means of rest and sedation for a fortnight. As there was only slight improvement, it was decided to prescribe methyl thiouracil. The initial dose of 0·3 gramme daily was increased after ten days' treatment to 0·4 gramme daily. In six weeks' time the patient showed considerable improvement. Her weight had increased by over a stone, the pulse rate had decreased to 78 per minute and the blood pressure readings had fallen to systolic, 140 millimetres of mercury, and diastolic, 90 millimetres. The basal metabolic rate was +6% and the serum cholesterol content was 245 milligrammes per 100 millilitres. At the time of the meeting the patient was receiving 0·3 gramme of methyl thiouracil daily, and it was anticipated that this dose would be reduced. It did not appear that operation would be necessary in this case.

The second patient was an unmarried woman, aged eighteen years. Her illness had commenced in December, 1948, when she had had an attack of tachycardia with breathlessness and faintness. Since then several similar turns had occurred. She had lost half a stone in weight during the past seven months. She was subject to severe headache. For four years she had performed strenuous work on her father's farm.

On examination of the patient on June 16, 1949, it was seen that there was moderate diffuse enlargement of the thyreoid gland, which was soft. There were no eye signs. There was no tremor of the hands. The pulse rate was 150 per minute and the blood pressure readings were as follows: systolic, 190 millimetres of mercury, diastolic, 100 millimetres. Her mother had been treated for thyreotoxicosis. The urine contained a small amount of glucose. The basal metabolic rate was +56% and the serum cholesterol content was 85 milligrammes per 100 millilitres.

After preliminary bed rest methyl thiouracil was prescribed. She made good progress for six weeks. Then she suffered from nausea and visual disturbances. Although the leucocyte count had not fallen, it was decided to suspend the treatment for a week. At the time of the meeting, therapy had been resumed and the patient was taking 0·3 gramme daily. Dr. Stokes remarked that the increased activity of the thyreoid gland would probably be controlled by methyl thiouracil without recourse to operative measures.

Deltoid Paralysis.

Dr. Stokes's next patient was a married woman, aged forty-nine years. She had first been examined on August 15, 1949. She stated that sixteen days previously she had experienced pain in the back of her neck, extending to her right shoulder. Two days after the onset of the pain she noticed loss of movement of her right arm.

On examination of the patient, wasting of the right deltoid region was obvious. The biceps, supraspinatus and infraspinatus muscles were affected to a lesser degree. She was unable to abduct the right arm at the shoulder and the movement of circumduction was impaired. No change was noted in sensory or reflex functions; in particular, the pupils were equal in size and reacted to light and accommodation and the knee jerks were active. X-ray examination of the right shoulder failed to reveal any bony abnormality. The muscles of the right shoulder girdle reacted normally to both faradic and galvanic current. A tattoo mark, representing a true lover's knot, was present near the tip of the left shoulder. In view of the presence of this mark, it was decided to examine the blood serum, which gave a complete positive result to the Wassermann test. However, the cerebro-spinal fluid gave no reaction to this test and in all other respects was normal.

A mixture containing potassium iodide, 10 grains, and *Liquor Hydrargyri Perchloridi*, one drachm, was prescribed to be taken three times daily. There was an almost immediate increase in the power of the right deltoid muscle. At the time of the meeting the patient was receiving intramuscular injections of "Rubyl" (3·5 millilitres) twice weekly and was also taking the mixture. The improvement in power and movement of the shoulder muscles was continuing, but the wasting, as was to be expected, still persisted. It was proposed to administer penicillin and neoarsphenamine shortly.

Clinical Photographs.

Dr. Stokes next showed a series of clinical photographs which included examples of mongolism, congenital syphilis, cretinism, thyreotoxicosis, simple goitre, myxœdema, acromegaly, nephritis, gouty tophi, ulceration of the tongue in Hodgkin's disease, lingual hemiatrophy, chancre of the upper lip, epithelioma of the lower lip, syphilitic ulceration of the palate, Paget's disease of bone, hemiplegia, *tabes dorsalis*, facio-scapulo-humeral dystrophy, *lymphogranuloma inguinale*, myelocythæmia, leprosy, sclerodactyly, clubbed fingers in bronchiectasis, wasting of the hands resulting from cervical rib, koilonychia, rheumatic nodules and deltoid paralysis. X-ray films of gastric carcinoma, of gastric ulcer, of colonic carcinoma, of intestinal obstruction by a band, of megacolon, of gall-stones and of pulmonary tuberculosis were also shown, together with electrocardiograms of right bundle branch block and auricular premature contractions and specimens of phæochromocytoma. The photographs were taken by Mr. L. W. Appleby, photographer to Sydney Hospital.

Phonocardiograms.

Dr. Stokes finally showed phonocardiograms illustrating a split first sound, mitral systolic murmurs, a presystolic murmur and protodiastolic gallop rhythm. He gave it as his opinion that apparatus on which phonocardiograms were recorded should be standardized so that pitch and volume could be measured. The phonocardiograms were taken by Dr. Ian Thomas and Dr. Warren Smith, medical associates to Sydney Hospital.

Bronchiectasis.

Cases of bronchiectasis and cystic disease were presented by the staff of the pulmonary clinic—DR. W. L. CALOV, DR. A. B. HOGAN, DR. M. F. DECK and DR. J. B. PHILLIPS. A demonstration of the physiotherapeutic measures employed in bronchiectasis was given by Miss Mitchell, of the department of physiotherapy.

Dr. Calov said that bronchiectasis of young people was one of the dangerous pulmonary diseases, frequently leading to death from sepsis or cerebral embolism in middle life or earlier. In many cases the patient's life was punctuated by periodic attacks of bronchopneumonia. In the intervals, the patient might be comparatively well except for cough and profuse sputum. In some cases, bouts of bronchopneumonia were rare or did not occur at all. In other cases, in which sepsis was great and large masses of lung were involved, breathlessness, cough and sputum were constant. It was remarkable how well many of those patients claimed to be and how well they appeared to be. But the outlook was not less grave for that. The essential treatment was surgical, and this was always advisable when it was practicable. When, for one reason or more, operation was impracticable, breathing and postural exercises sometimes gave considerable help. Many patients also received temporary benefit from bronchoscopic suction. Some of the patients at the clinic attended regularly for the direct instillation of a penicillin solution into the trachea. It was, as yet, impossible to assess the value of that measure in the light of their experience, which had been too short. Some patients took regular small doses of sulphanilamide. The main object in that was the prevention of bronchopneumonia. That kind of therapy had a logical basis. Whether it would prove of practical value remained to be seen.

The first patient shown was a man, aged fifty-seven years, who had suffered from a cough with sputum for three or four years. He had an hæmoptysis after swallowing a fish bone nine months prior to the meeting. At that time he was radiologically examined and was considered to be suffering from pulmonary tuberculosis. He had been in a sanatorium for the past six months, had suffered from attacks of asthma for four years and had lost three stone in weight in three years. At an X-ray examination on February 24, 1949, the possibility of cystic disease of the lung could not be excluded. A bronchogram was prepared and showed several large cystic areas in the upper lobe of the right lung and in the base of the lower lobe of that lung. No tubercle bacilli were detected in the sputum. The Mantoux test produced a positive reaction.

A female patient, aged thirty-six years, was then shown. She had suffered from pneumonia at the age of two years, followed by the appearance of bronchiectasis. She had had frequent attacks of pneumonia during the last three years. At bronchoscopy copious purulent sputum was aspirated from both stem bronchi. Culture of the sputum produced a growth of *Streptococcus viridans* but no tubercle bacilli. X-ray examination of the chest revealed bronchiectasis at the base of the right lung. Bronchography revealed extensive bilateral bronchiectasis. In treatment sulphonamides and penicillin had been used, with bronchoscopic drainage and postural drainage.

A male patient, aged forty years, was also shown. In 1937, at the age of twenty-eight years, he had suffered an attack of pleurisy and was found to have tuberculosis. He was treated at Randwick Auxiliary Hospital and later at Queen Victoria Sanatorium. Artificial pneumothorax was induced in 1937. This was followed by phrenic crush. He attended Sydney Hospital first in 1940, shortly after his discharge from the sanatorium. At that time he had a cough and some sputum. His weight was a little below normal. X-ray examination revealed appearances indicative of extensive fibro-caseous tuberculosis of the left lung. Tubercle bacilli were not found in the sputum. His blood sedimentation rate was six millimetres in one hour. He was advised to continue the sanatorium régime at home.

Hæmoptysis occurred in 1944. Tubercle bacilli were recovered from the sputum. Cavitation appeared in the left subapical region. He was admitted to hospital under the care of Dr. M. P. Susman. Thoracoplasty was performed in four stages. The first six ribs and the posterior parts of the seventh and eighth ribs were removed. He then spent twelve months in a sanatorium and progressed favourably.

His condition continued to be satisfactory until January, 1948, when he complained of sweating and breathlessness. At this time he was living on a poultry farm and appeared to be working too hard. In February, 1948, he coughed blood. His pulse rate was 120 per minute. He looked ill, and was breathless at rest. In June, 1948, he was admitted to hospital and given a course of 37 grammes of streptomycin. The ribs as far as the eleventh were removed by Dr. Susman.

From December, 1948, to March, 1949, he was again in a sanatorium.

At the time of the meeting the patient still had a cough with about two ounces of sputum per day. A bronchogram showed complete collapse of the left lung with bronchiectasis. It was pointed out that that was a not uncommon result of thoracoplasty. The bronchiectasis explained the cough and sputum that persisted in some cases. It was of interest to note in the case under discussion that the patient had a chronic antrum infection.

A female patient, aged eighteen years, suffering from bilateral bronchiectasis, was also shown. The patient had presented in February, 1948, with the complaints of pain in the left side of the chest of three days' duration, and cough of three months' duration. For the past nine years, since the age of nine years, the patient had had a cough, which was usually mild, but which had become more pronounced in the last three months. She now produced half a cup of fœtid creamy sputum per day, mostly in the mornings. She had had a stabbing pain over the left lower ribs for one week, which had been continuous for three days. Her appetite was poor and she tired easily. There were no symptoms referable to other systems. The patient had suffered from chronic sinusitis since the age of nine years. She had had two operations on the nose and antra at the ages of nine and fourteen years. She had undergone two tonsillectomies at the ages of four and ten years. All her top teeth had been removed three years prior to the meeting.

On examination of the patient she was in a good state of nutrition and not pale. Early clubbing of the digits was present. There were numerous rhonchi and râles at both lung bases, but no other abnormality was detected in the lungs.

A bronchographic examination disclosed bilateral saccular bronchiectasis. The lingula of the upper lobe of the left lung was involved. The right middle lobe bronchi appeared regular. Bronchoscopic examinations on various occasions showed pus coming from the lower stem bronchi, sometimes only from the left, from which there was also some hæmorrhage on one occasion. The mucous membrane was reddened and inflamed. Tests of the sputum failed to reveal tubercle bacilli. The Mantoux test produced a "doubtful positive" response. A blood count revealed no anæmia, but slight neutrophilia (9039 neutrophile cells per cubic millimetre).

The patient was treated with postural drainage twice daily, and she had also been taught breathing exercises. Bronchoscopic drainage had been carried out at regular intervals, and was now performed monthly. The patient said that she felt better for the drainage, and was at the time of the meeting in good general health.

The comment was made that the onset of the symptoms of the disease in this case at the age of nine years, at the same time as it was discovered that the patient had a chronic sinus infection, was interesting, as it pointed to the importance of one of the well-known causative factors in bronchiectasis—namely, the inhalation of infective material from the upper respiratory tract, which it was now known could readily occur during sleep, anæsthesia *et cetera*. The symptomatology in the case under discussion was typical of bronchiectasis of moderate severity. With regard to treatment, it was pointed out that the treatment of the sinus infection was highly important. The patient had the routine treatment of postural drainage and breathing exercises. The efficacy of repeated bronchoscopic drainages was controversial. A cure would involve the removal of both lower lobes and the lingula of the upper lobe of the left lung. Such extensive lung surgery had been performed, and, it had been asserted, no great respiratory distress resulted. The danger of the development of severe emphysema or bronchiectasis in the remaining lung tissue at a later date had to be considered. On the other hand, removal of the diseased lung tissue on the worse affected side (in the present case, the left) had been advocated, as it had been shown in many cases to relieve a patient of the unpleasant symptoms to such an extent that further surgery had not been wanted or considered necessary.

A female patient suffering from early bronchiectasis was then shown. The patient had presented herself in August, 1949, complaining of having had a cough all her life and of tiredness of two months' duration. She suffered from almost continuous colds. The cough was worse in the mornings and was productive of one ounce of white or yellow sputum per day, which was not offensive, but had been blood-stained on two occasions. She had had a pain in the left side of her chest for two months which was diagnosed as pneumonia. She was somewhat breathless on exertion. She complained of a sore throat and of slight hoarseness. She tired easily and slept poorly, but her appetite was good. She had no symptoms referable to other systems. She had suffered from measles and varicella as a child and from mumps at the age of eighteen years. She had undergone tonsillectomy three times at the age of twelve years. The patient had three healthy children, but her father suffered from bronchitis. She did not smoke tobacco or drink alcoholic liquor.

On examination, the patient was of good physique and in a good state of nutrition. She had a healthy appearance. There was no tachycardia and her tongue was clean. No clubbing of the digits was present and the fauces were clear. There was no tenderness over the sinuses. The chest had good expansion; an occasional rhonchus was heard, but no other abnormality was detected in the lungs. The cardiovascular system, the genito-urinary system and the central nervous system all appeared normal. A bronchographic examination on August 30, 1949, revealed irregularity and some dilatation of the anterior and middle basal branches of the right lower lobe bronchus, and slight irregularities in some of the bronchi of the left lung. It was considered that the lesions might be congenital. A bronchoscopic examination on the same date revealed very little secretion from the left main bronchus only. No tubercle bacilli were detected in the sputum and the Mantoux test produced no reaction. The blood sedimentation rate was four millimetres (Westergren method). At the time of the meeting the patient was learning breathing exercises; she had had only one bronchoscopic drainage.

The comment was made that several facts suggested the importance of a congenital factor in the causation of the disease in the case under discussion—namely, the onset in childhood, the family history of bronchitis and the X-ray findings of congenital abnormalities of bronchi of the contralateral lung. The symptomatology was typical of mild bronchiectasis. The treatment in the case under discussion raised two questions: (i) whether, in view of the possible congenital basis, there might not develop after a postulated right lower lobectomy further bronchiectasis in other parts of the lung; (ii) whether in a case in which the symptoms were so mild and the patient was in such good general health, the manifest severity and risks of radical operation were justified. Against those questions must be weighed the fact that localized bronchiectasis was known to progress and extend, and also that there existed risks of complications such as pneumonitis, pulmonary fibrosis and collapse, cerebral abscess *et cetera*.

Pulmonary Tuberculosis.

A male patient, aged forty-five years, was shown by the staff of the pulmonary clinic. The patient had first reported in March, 1940, with the history that he had been discharged from a sanatorium one week previously. X-ray examination revealed fibrotic tuberculosis at the apex of the right lung, with some cavitation and slight increase in markings of the middle zone of the left lung. Acid-fast bacilli were present in the sputum. The patient was observed with no great change, but on July 23, 1940, he had a small hæmoptysis. He was not examined again until June, 1945, when an X-ray examination revealed practically no change. He was again examined in June, 1946, when an X-ray examination revealed no change. He had another hæmoptysis in August, 1946, and in October, 1946, he underwent right phrenic crush with good paralysis and elevation of the diaphragm. Acid-fast bacilli were present in the sputum. The comment was made that the patient had held his condition well, but the cavity had become a little larger. Acid-fast bacilli were still present in the sputum, the left lung was now clear and he was recommended for thoracoplasty.

Diabetes Mellitus with Arterial Degeneration.

DR. W. L. CALOV showed a female patient, aged seventy years, suffering from *diabetes mellitus* with arterial degeneration. A diagnosis of *diabetes mellitus* had been made in 1930. She was treated by diet alone until 1939, when insulin was given. Her diabetes was fairly well controlled with small doses of soluble insulin and later with protamine zinc insulin. Her weight was maintained. In February, 1943, a small ulcer appeared on the right great toe. This was kept in check for about six months, when a rapid spread of the gangrene occurred with sepsis. The limb was amputated through the thigh in August, 1943. From time to time since then she had complained of paræsthesiæ of the left leg and the fingers. From time to time also she had had small septic lesions of the toes. The pulsation of the *dorsalis pedis* artery was readily palpable. X-ray examination revealed no calcification of the arteries. At the same time it was apparent that degenerative changes had taken place in the arteries of the lower limb. Changes in the toes and nails were apparent as a result of impairment of the blood supply.

Rehabilitation Service.

The almoner's department presented a demonstration to draw attention to the objects and scope of a rehabilitation service for hospital patients. It was pointed out that the purpose of rehabilitation, according to the report of the Baruch Committee on Physical Medicine, was "to prepare the patient physically, mentally, socially and vocationally for the fullest possible life compatible with his abilities and disabilities". Medical treatment and appropriate nursing care were implicit in the rehabilitation process; but the term was used to describe "any process other than medical and surgical treatment applied to an individual to prevent or reduce his loss of capacity and to restore him to the maximum possible degree of mental and physical ability in the shortest possible time after illness or injury and his reinstatement into constructive work as a useful and contented member of the community" (Dr. P. J. Macleod). The rehabilitation process services included the following: (i) physical and mental reconditioning—physical education, physiotherapy and occupational therapy; (ii) social study and guidance; (iii) financial assistance; (iv) vocational guidance and training; (v) reemployment—in normal industry, in sheltered workshops and at home. Such services might be helpful to many different kinds of patients, for example: (i) the physically handicapped child or young person whose education or choice of employment was affected by his handicap; (ii) the disabled adult who had to change his occupation; (iii) the aging unskilled worker who was no longer fit for laborious work; (iv) the patient whose mental and physical vigour was endangered by prolonged inactivity.

Rehabilitation centres might be established in a community by several different methods: (i) by official action of a general assembly, a county board, a city council or similar public body; (ii) by industrial or labour groups, manufacturers' associations, chambers of commerce, unions *et cetera*; (iii) by social agencies of various types; (iv) by any combination of the above-mentioned groups organized into a joint board or committee; (v) by medical schools or hospitals. Each of the methods mentioned had been successfully used for different purposes. For example, a rehabilitation centre attached to a hospital accomplished the following: (i) enabled in-patients and out-patients to participate in a rehabilitation programme at the earliest appropriate stage in treatment; (ii) ensured continuity of supervision of the patients by the hospital medical staff; (iii) provided for

effective cooperation between members of the hospital rehabilitation team and services provided by governmental or other agencies in the community; (iv) offered to practitioners and students in medicine and ancillary professions opportunities for training and experience in principles and methods of rehabilitation.

It was pointed out that the Australian Government was empowered to develop and finance, and was in process of developing, a rehabilitation service in Australia; this service was being offered to certain groups of ill and disabled persons including public hospital patients. Rehabilitation centres were regarded as an essential part of the service, and some experimental centres were already established and administered by the Government. As many of the patients referred to almoners by members of the medical staffs of hospitals needed the help of a rehabilitation service while still in the care of the hospital, cooperation between the hospitals and government rehabilitation centres was essential. Several questions had to be considered in the application of such a service to hospital patients: (i) Should public hospitals in Australia provide rehabilitation centres for their patients? (ii) Would it be possible and desirable to seek assistance from the Commonwealth Government for that purpose? (iii) What form should a hospital rehabilitation centre take—what services should it offer and how should it be administered?

A short outline of ten cases was finally presented, to illustrate difficulties which beset some hospital patients, and in which help might be given by a rehabilitation centre.

Correspondence.

THE WORLD PROBLEM AND PSYCHIATRY.

SIR: Your leading article on "The World Problem and Psychiatry" (THE MEDICAL JOURNAL OF AUSTRALIA, October 8, 1949, page 543) deserves at the very least one letter supporting it. I would like to refer to two points: one which you made and one which you omitted. You state that it is open to doubt whether appeals for help made to the psychiatrist in other places by professional people are common in Australia. One of the factors which prevent people from appealing to the social psychiatrist (and Professor McDougall's book on "Social Psychology" fully justifies the term) is the fact that they rely too much upon their own introspective psychology. Introspective psychology is probably the least informative and the most unreliable of all form of practical psychology. Yet how often does one hear even a doctor minimize a clinical syndrome because "I have done that myself for years"!

The second aspect of "cooperative effort" and "social solidarity" to which I would refer is housing. No scheme of community psychological readjustment can succeed unless it includes a programme of housing which is producing results. In this country I do not think it is appreciated that the science of housing is a medical one. Here your housing departments are associated with brick production and the timber industry. This is wrong. Housing should be a subsection of the health department as it is in Britain. This is no idle claim by me, for those of your readers who have read Aristotle will remember how often even thousands of years ago he associated health with housing. This relationship existed in his very clear mind only as a philosophical conclusion. But practical experience has made it clear all over the world that if the medical profession is not associated with housing it becomes a social liability rather than an asset.

Yours, etc.,

65 Roseberry Street, Bedford Park, Perth.
October 19, 1949.

JOHN A. MCCLUSKIE.

AN APPEAL.

SIR: I beg to ask for the hospitality of your columns in order to appeal to your readers for contributions towards the funds of the Princess Tsahai Memorial Hospital.

The genesis of this hospital is as follows. The Emperor of Ethiopia, Haile Sellassie, was driven from his country by the armies of Mussolini, who did not hesitate to order the employment of gas against the defenceless Ethiopians. "This isn't war, it's torture of tens of thousands of defenceless men, women and children, with bombs and poison gas"—so wrote the head of the British Ambulance Service.

Princess Tsahai, a girl of seventeen, came into sad exile in Britain, and at once sought work as a probationer nurse, so that she might be able to serve one of her country's greatest needs when liberation came, as she never doubted it would. She was not very strong, and a nurse's training is hard, but she had all the qualities of a good children's nurse—patience, gaiety of heart, gentleness, self-control and love. The children loved her, "they were sweet and understanding", she wrote to a friend. Her matron there wrote: "Guy's Hospital will remember her as one of whom it will always be proud." She was at Guy's when it was terribly bombed during the war.

Ethiopia was liberated, and Princess Tsahai returned to her country, after five years' service as a nurse in Britain, to find friends and relatives dead, thousands of homeless, maimed and wounded, thousands of orphaned children thronging the streets. She opened a home for the orphans, clinics and dressing stations, giving her knowledge and all her strength to the service of the sufferers. Then she married, and went with her husband to a remote district, where she at once opened a dressing station, and had dreams of a hospital, but working too hard, she was struck down and died at the age of twenty-two, lacking the skilled help she had devoted herself to bringing to others.

It was to commemorate the life of this good and sweet girl that some of us decided to build a hospital in Addis Ababa to her memory—a hospital with wards for children and maternity, and provision also for teaching and training. We set out to raise £100,000 and have obtained £85,000 of that sum. The hospital is built, and partly equipped; we want £15,000 to complete the enterprise. I appeal to your readers to help us to this end. I do so confidently because I know the object is a good one, and also those who give will be doing something to show in a foreign and backward country that the English-speaking race believes in mercy, pity and loving kindness, the qualities of which a hospital is the finest possible outward and visible sign.

Donations should be sent to the Honorary Treasurers, Lord Horder and Lord Amulree, c.o. Messrs. Gould and Prideaux, 88 Bishopsgate, London, E.C.2, the Honorary Accountants, who will gratefully acknowledge receipt.

Yours, etc.,

[LORD] WINSTER, Chairman.

London,
September 19, 1949.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, promotions *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 76, of October 20, 1949.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Medical Branch.

The appointment of Flight Lieutenant P. M. Birrell (297470) is terminated on demobilization, 11th October, 1949.

Reserve: Medical Branch.

The following are appointed to commissions, 26th August, 1949, with the rank of Flight Lieutenant: John King Joyce (277559), John Dudley Ward (277560).—(Ex. Min. No. 49—Approved 13th October, 1949.)

Congresses.

A CONFERENCE OF MEDICAL WAR HISTORIANS.

THE third meeting of the medical war historians of the Commonwealth countries was held at Canberra from October 3 to 8, 1949. A liaison committee was formed in 1946, as described recently in a special article by W. Franklin Mellor in this journal, including the official medical historians of the United Kingdom, of Australia, of Canada, of India, of New Zealand and of South Africa, and latterly of the United States of America. The members attending the conference at Canberra were as follows: Professor F. A. E. Crew, Squadron-Leader H. N. H. Genese and W. Franklin

Mellor (Secretary), of the United Kingdom; Dr. Allan S. Walker, of Australia; Dr. W. R. Feasby, of Canada; and Dr. T. D. M. Stout, of New Zealand. Observers were also present as follows: representing the Royal Australian Navy, Surgeon Captain D. A. Pritchard and Mr. H. W. Martin; representing the Royal Australian Air Force, Air Commodore E. A. Daley; representing the Combined Historical Section of India, Mr. J. N. Dhamija; and representing the United States Histories, Mr. P. Jarman, the Ambassador. Dr. Allan S. Walker was elected chairman.

Full sessions were held on part of each day, and for the remainder of the time, extending into the following week, sectional discussions were held covering subjects of joint interest to various groups. In this way questions of procedure and policy were debated and decided, and details of administration, and of campaigns in which more than one country participated, were compared and corrected. A great amount of draft material was presented and exchanged, and important sections were read and examined. In this way overlapping is being minimized, a fuller and more accurate story is being compiled, and at least the same degree of cooperation and mutual help is being continued as during the years of war.

By agreement the clinical volumes have been the subject of concentrated work; most of these are nearing completion in draft, and many of these drafts have been exchanged.

The administrative histories are being compiled, and as much of these is peculiar to each country they should now proceed smoothly. The campaign stories have the next priority. Here the value of collaboration cannot be overestimated. It is hoped that within the next two years campaign stories will be circulated to the partners who are concerned in them, and that a worthy and accurate history will thus be presented.

A number of resolutions were submitted to the committee at the final session, at which representatives of all countries concerned, except South Africa, were present. Reaffirmation was made of the need for speedy production and circulation of draft narratives, in order to accelerate publication, and of the consequent need of adequate assistance from the authorities concerned. It was agreed that the Commonwealth country chiefly responsible for the work of any group or joint activity should undertake its recording. The value of well-founded criticism in the histories was also reaffirmed. It was regretted that the historians of several countries could not be present in person; the need of a further meeting at a later date was expressed in order to complete discussion and correction of campaign stories.

The Government of the Commonwealth of Australia, which had extended an invitation to the committee to meet at Canberra, showed practical interest in the work, and afforded all facilities necessary. Opportunity was given for all the delegates to meet representative senior members of the Government at luncheon, and through literature and excellent documentary films the visitors were able to learn much about Australia in the time available.

All the medical historians look to their respective governments and to their professional colleagues for adequate assistance in a task of great magnitude and importance.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Public Lecture by Professor F. A. E. Crew.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that Professor F. A. E. Crew, F.R.S., D.Sc., Ph.D., M.D., F.R.C.P. (Edinburgh), will give a public lecture on "Biological Aspects of Marriage" on Wednesday, November 9, 1949. The lecture will be held in the Stawell Hall, 145 Macquarie Street, Sydney, at 8.15 p.m. Tickets will be 5s. and the proceeds will be for the purchase of food parcels for Britain. Attendance is strictly limited and the list will be closed as soon as the necessary tickets have been sold. Applications, together with remittance, should be addressed to the Secretary of the Committee, 131 Macquarie Street, Sydney. Telephones: BU 5238-BW 7483.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED OCTOBER 15, 1949.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Ankylostomiasis	*	1	..	..	*	..	..	..	1
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	4(3)	..	1	..	..	..	..	..	5
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	*	*	..	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	1	..	..	..	..	..	1
Diphtheria	6(5)	7(3)	3(1)	..	2	..	..	..	18
Dysentery(*b*)	*	2	2	..	..	..	..	1	5
Encephalitis Lethargica	..	..	..	..	..	..	..	..	..
Erysipelas	*	*	*	4(2)	*	*	..	..	4
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	*	*	*	..	*	*	..	..
Influenza	*	*	*	..	*	*	..	*	..
Leprosy	..	..	..	..	..	..	..	..	..
Malaria(*c*)	*	1(*c*)	1(*c*)	(*c*)	(*c*)	(*c*)	(*c*)	(*c*)	2(*c*)
Measles	*	*	*	79(44)	*	*	..	..	79
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	4(1)	15(7)	2	32(31)	1	2(1)	..	..	56
Psittacosis	*	..	*	..	*	*	*	*	..
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella(*h*)	*	*	8(7)	*	4	*	..	..	12
Scarlet Fever	23(12)	17(6)	3(2)	14(6)	4	2(1)	..	1	64
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	..	*	*	*	*	*	..
Trachoma	*	..	*	*	*	*	*	*	..
Tuberculosis(*d*)	35(30)	26(13)	10(7)	2	16(4)	8(2)	..	..	97
Typhoid Fever(*e*)	..	..	..	..	..	..	..	..	..
Typhus (Endemic)(*f*)	..	..	1	..	1	..	..	..	2
Undulant Fever	..	1	..	..	..	..	..	..	1
Weil's Disease(*g*)	*	*	*	*	*	*	*	*	..
Whooping Cough	*	*	*	33(9)	*	*	..	..	33
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 36, 1944–1945. Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from Northern Territory.
* Not notifiable.
(*a*) Includes "Mossman" and "Sarina" fevers. (*b*) Includes amœbic and bacillary. (*c*) Statistics inexact with varying practice with regard to relapses in Service cases infected overseas. (*d*) Includes all forms except in Northern Territory, where only pulmonary tuberculosis is notifiable. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Cases reported include scrub, murine and tick typhus. (*g*) Includes leptospiroses, Weil's and para-Weil's disease. (*h*) Notifiable disease in Queensland in females aged over fourteen years.

Courses for Part II of the M.S., D.G.O., D.L.O. and D.O. Examinations.

Courses for Part II of the M.S., D.G.O., D.L.O. and D.O. will begin in Sydney on January 9, 1950, and continue for periods of twelve weeks. The fee will be £31 10s. for attendance at each course, and early enrolment with the Course Secretary is desirable.

Course for M.R.C.O.G. Examination.

The course for Part II of the D.G.O., which begins on January 9, 1950, will be suitable for candidates studying for the M.R.C.O.G. examination. In addition, however, candidates will receive advanced instruction applicable to the M.R.C.O.G. examination. Early enrolment with the Course Secretary is requested.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Fulde, Ewald Adolf Oscar, registered in accordance with the provisions of Section 17(2) of the *Medical Practitioners Act*, 1938-1945, 128 Brighton Boulevarde, North Bondi.

Australian Medical Board Proceedings.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1939 *to* 1948, of Queensland, as duly qualified medical practitioners:

Mair, John Russell, M.B., B.S., 1948 (Univ. Sydney), c.o. Hospitals Board, Rockhampton.

Stuckey, Gordon Clarence, M.B., B.S., 1948 (Univ. Sydney), Brisbane General Hospital, Brisbane.

Macken, Faith Marion, M.B., B.S., 1934 (Univ. Sydney), Peel Island Lazaret, via Brisbane.

McDermott, George Lewellyn, L.M.S.S.A., 1928 (Univ. London), Aramac.

Wiles, Helen Booth, M.B., B.S., 1945 (Univ. Sydney), c.o. District Hospital, Herberton.

The following additional qualification has been registered:

Harrison, Andrew, 97 Wickham Terrace, Brisbane, D.O.M.S., R.C.P. and S. (England), 1948.

Corrigendum.

IN the paper "The Treatment of Acute Lumbago and Acute Low-Back Strain in General Practice" by James H. Young in the issue of October 22, 1949, "cannot" in line 7 of page 597 should read "can seldom" and "81" in line 9 of page 599 should read "481". We regret these errors.

Obituary.

JOHN HAMILTON CRAWFORD, JUNIOR.

WE regret to announce the death of Dr. John Hamilton Crawford, junior, which occurred on October 7, 1949, at Pomona, Queensland.

FRANCIS SYNDAL COOMBS.

WE regret to announce the death of Dr. Francis Syndal Coombs, which occurred on October 9, 1949, at Numurkah, Victoria.

DONALD LUKER.

WE regret to announce the death of Dr. Donald Luker, which occurred on October 22, 1949, at Katoomba, New South Wales.

Diary for the Month.

Nov. 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Nov. 9.—Victorian Branch, B.M.A.: Branch Meeting.
Nov. 10.—New South Wales Branch, B.M.A.: General Meeting.
Nov. 10.—Victorian Branch, B.M.A.: Organization Subcommittee.
Nov. 11.—Queensland Branch, B.M.A.: Council Meeting.
Nov. 14.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.
Nov. 15.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Nov. 16.—Western Australian Branch, B.M.A.: General Meeting.
Nov. 17.—New South Wales Branch, B.M.A.: Clinical Meeting.
Nov. 17.—Victorian Branch, B.M.A.: Executive Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135 Macquarie Street, Sydney): Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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